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EPONYMS.

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VIII. POTT'S FRACTURE.

THE fact that Pott broke his leg and also wrote about fractures of the leg has led many to believe that he himself suffered from the injury which is now called 'Pott's fracture'. It is clear, from the account given by his son-in-law, Sir James Earle, that his accident was a compound fracture of the tibia. He led an active and useful life for many years afterwards, so that it is probable the fibula was not broken, and that union took place with good alinement.

Writing in 1768, twelve years after his own accident, he says in his *Remarks on Fractures*:—

"Whoever will take a view of the leg of a skeleton, will see that although the fibula be a very small and slender bone, and very inconsiderable in strength when compared with the tibia, yet the support of the lower joint of that limb (the ancle) depends so much on this slender bone, that without it the body would not be upheld, nor locomotion performed, without hazard of dislocation every moment. The lower extremity of this bone, which descends considerably below that end of the tibia, is by strong and inelastic ligaments firmly connected with the last-named bone, and with the astragalus, or that bone of the tarsus which is principally concerned in forming the joint of the ancle. This lower extremity of the fibula has, in its posterior part, a superficial sulcus for the lodgement and passage of the tendons of the peronei muscles, which are here tied down by strong ligamentous capsulæ, and have their action so determined from this point or angle, that the smallest degree of variation from it, in consequence of external force, must necessarily have considerable effect on the motions they are designed to execute, and consequently distort the foot. Let it also be considered, that upon the due and natural state of the joint of the ancle, that is, upon the exact and proper disposition of the tibia and fibula, both with regard to each other and to the astragalus, depend the just disposition and proper action of several other muscles of the foot and toes; such as the gastrocnemii, the tibialis anticus, and posticus, the flexor pollicis longus, and the flexor digitorum pedis longus, as must appear demonstrably to any man who will first dissect, and then attentively consider these parts.

"If the tibia and fibula be both broken, they are both generally displaced in such manner, that the inferior extremity, or that connected with the foot, is drawn under that part of the fractured bone which is connected with the knee; making by this means a deformed, unequal tumefaction in the fractured part, and rendering the broken limb shorter than it ought to be, or than its fellow. And this is generally the case, let the fracture be in what part of the leg it may.

"If the tibia only be broken, and no act of violence, indiscretion, or inadvertence be committed, whether on the part of the patient or of those who conduct him, the limb

most commonly preserves its figure and length; the same thing generally happens if the fibula only be broken, in all that part of it which is superior to letter A in the annexed figure, (*Fig.* 333) or in any of it between its upper extremity, and within two or three inches of its lower one.

"I have already said, and it will obviously appear to every one who examines it, that the support of the body, and the due and proper use and execution of the office of the joint of the ancle, depend almost entirely on the perpendicular bearing of the tibia upon the

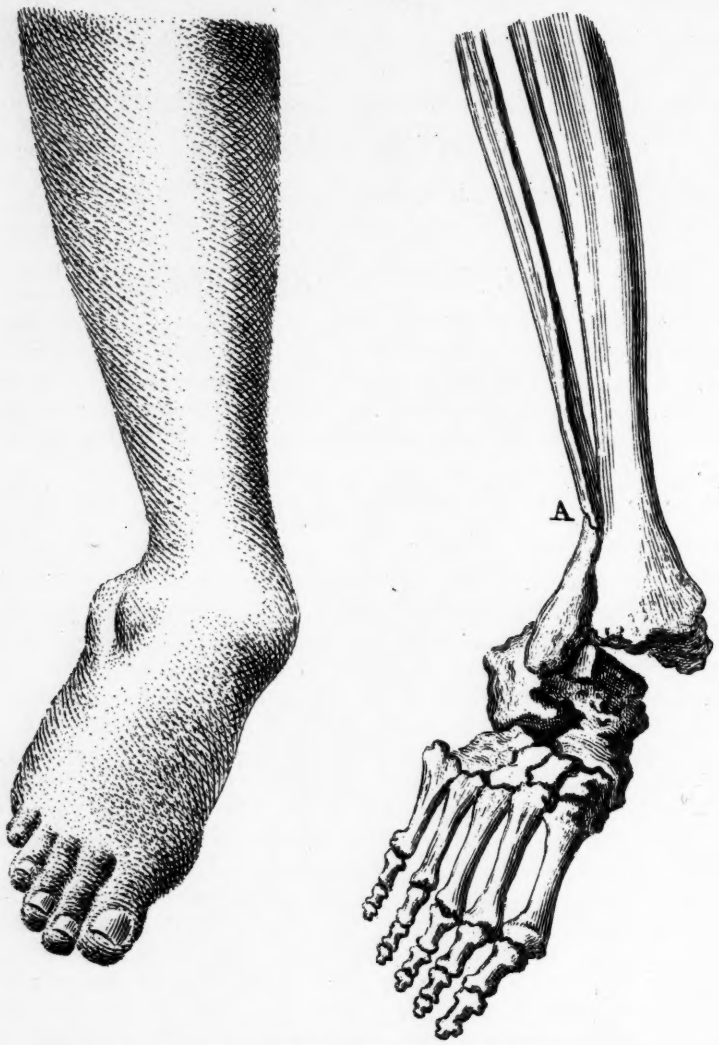

FIG. 333.—From the original illustration of Pott's fracture.

astragalus, and on its firm connection with the fibula. If either of these be perverted or prevented, so that the former bone is forced from its just and perpendicular position on the astragalus; or if it be separated by violence from its connection with the latter, the joint of the ancle will suffer a partial dislocation internally *; which partial dislocation cannot happen without not only a considerable extension, or perhaps laceration of the bursal ligament of the joint, which is lax and weak, but a laceration of those strong tendinous ligaments, which connect the lower end of the tibia with the astragalus and

* See the figure.

os calcis, and which constitute in great measure the ligamentous strength of the joint of the ancle.

"This is the case, when, by leaping or jumping, the fibula breaks in the weak part already mentioned, that is within two or three inches of its lower extremity. When this happens the inferior fractured end of the fibula falls inward toward the tibia, the extremity of the bone which forms the outer ancle is turned somewhat outward and upward, and the tibia having lost its proper support, and not being of itself capable of steadily preserving its true perpendicular bearing, is forced off from the astragalus inwards, by which means the weak bursal, or common ligament of the joint is violently stretched, if not torn, and the strong ones, which fasten the tibia to the astragalus and os calcis, are always lacerated; thus producing at the same time a perfect fracture and a partial dislocation, to which is sometimes added a wound in the integuments, made by the bone at the inner ancle. By this means, and indeed as a necessary consequence, all the tendons which pass behind or under, or are attached to the extremities of the tibia and fibula, or os calcis, have their natural direction and disposition so altered, that instead of performing their appointed actions, they all contribute to the distortion of the foot, and that by turning it outward and upward.

"When this accident is accompanied, as it sometimes is, with a wound of the integuments of the inner ancle, and that made by the protrusion of the bone, it not infrequently ends in a fatal gangrene, unless prevented by timely amputation, though I have several times seen it do very well without. But in its most simple state, unaccompanied with any wound, it is extremely troublesome to put to rights, still more so to keep it in order, and unless managed with address and skill, is very frequently productive both of lameness and deformity ever after.

"After what has been said, a further explanation why this is so is unnecessary. Whoever will take even a cursory view of the disposition of the parts, will see that it must be so. By the fracture of the fibula, the dilatation of the bursal ligament of the joint, and the rupture of those which should tie the end of the fibula firmly to the astragalus and os calcis, the perpendicular bearing of the tibia on the astragalus is lost, and the foot becomes distorted; by this distortion the direction and action of all the muscles already recited are so altered, that it becomes (in the usual way of treating this case) a difficult matter to reduce the joint, and, the support of the fibula being gone, a more difficult one to keep it in its place after reduction. If it be attempted with compress and strict bandage, the consequence often is a very troublesome, as well as painful, ulceration of the inner ancle, which very ulceration becomes itself a reason why such kind of pressure and bandage can be no longer continued; and if the bone be not kept in its place, the lameness and deformity are such as to be very fatiguing to the patient, and to oblige him to wear a shoe with an iron or a laced buskin, or something of that sort, for a great while, or perhaps for life.

"All this trouble, pain, difficulty and inconvenience are occasioned by putting and keeping the limb in such a position as necessarily puts the muscles into action, or into a state of resistance, which in this case is the same. This occasions the difficulty in reduction, and the difficulty in keeping it reduced; this distorts the foot, and by pulling it outward and upward makes that deformity, which always accompanies such accident; but if the position of the limb be changed, if by laying it on its outside, with the knee moderately bent, the muscles forming the calf of the leg, and those which pass behind the fibula and under the os calcis, are all put into a state of relaxation and non-resistance, all this difficulty and trouble do in general vanish immediately; the foot may easily be placed right, the joint reduced, and by maintaining the same disposition of the limb, everything will in general succeed very happily, as I have many times experienced."

This account of fractures of the leg and their treatment gives a fair example of the style and methods of Percivall Pott's teaching. It shows him to have been a clear and logical thinker who based his knowledge partly upon anatomy and partly upon the results of his own experience.

HYPERPLASIA OF EPITHELIAL AND CONNECTIVE TISSUES IN THE BREAST: ITS RELATION TO FIBRO-ADENOMA AND OTHER PATHOLOGICAL CONDITIONS.

By Sir GEORGE LENTHAL CHEATLE, London.

I WANT the reader to bear in mind that, however great the prominence given in this article to the hyperplasia of the connective tissues, the epithelial changes may be predominant in all tumours where epithelial elements are concerned. Although the hyperplasia of epithelium of the breast may be the predominant factor in tumour formations, or hyperplasia of epithelial and connective tissues may be mutually correlated in that process, the morphological history of the breast should be borne in mind to this extent, that had it not been for the primary dip down of surface epithelium to form the secreting elements of the gland, the connective-tissue elements would have been absent.

Another point to which I must draw attention is this, that morphologically the epithelium lining ducts and acini is really an external tissue derived from the epiblast. To describe the epithelium of ducts and acini as 'external' seems too pedantic, and in all my description I regard the epithelium as being the most internal layer.

To clarify the following description I draw attention to *Fig.* 334, which represents diagrammatically the anatomical structures constituting the ducts and acini: (A) The terminal duct; (B) and (B_1) Acini; (C) The epithelium of the duct; (D) The epithelium of the acini.

Immediately underneath the epithelium there is a single layer of longitudinally arranged unstriated muscle fibre (E), which lies on the basement membrane (F), underneath which is a layer of delicate connective tissue consisting of bipolar and stellate cells (G).

The elastica (H) is seen outside the delicate connective tissue G. This relation of the elastica is maintained in ducts and acini. A very few lobules of the gland have a fine layer of elastica surrounding them, but this is very rare. Although the elastica surrounds a great many acini, it cannot be demonstrated to surround all acini even in the normal breast. (*See* Acinus B_1, *Fig.* 334.) By a normal breast I mean the average breast of 20 years of age. Sections of whole breasts examined after the age of 35 show that many glands from this age upwards cannot really be regarded as being in the normal state.

It is important to know that, whether the elastica be continued from the duct to surround the acini or not, there is no doubt that the tissues E, F, and G of the duct are always in direct continuity with the identical tissues that surround the acini. (*See Fig.* 334, B_1, and also *Fig.* 348.)

(I) Represents the unstriated muscle in the duct wall outside the elastica, which gradually gets less, and disappears when the acini are reached.

The walls of ducts and individual acini are all very closely invested by fibrous tissue (K), which forms, as it surrounds the acini, the intralobular connective tissue (K_1). This fibrous tissue which so closely invests the glandular structures appears to me to be of a more separate and specialized type than that which is diffused generally throughout the breast (L). I believe this to be true for the following reasons. It (K and K_1) varies in density where no variations occur in the general fibrous tissue, and it may remain unaffected when the general fibrous tissue of the breast appears to have undergone condensation. It degenerates where no degeneration is seen in the general fibrous tissue of the gland. When undergoing degeneration it stains differently from the surrounding fibrous tissue of the gland. It shares in a very marked degree in the hyperplasia of the elastica

when that event occurs (*Figs.* 350 and 351). Finally, it is primarily concerned in the formation of the peri-acinous and pericanalicular variety of fibro-adenoma, and also some forms of multiradicular papillomata.*

I divide the hyperplasia I am about to describe into three classes : (I) *The hyperplasia intra-elastica* ; (II) *The hyperplasia elastica* ; (III) *The hyperplasia extra-elastica.* These classes have a special bearing on localized and diffused fibro-adenomatous conditions, and also upon the formation of papillomata.

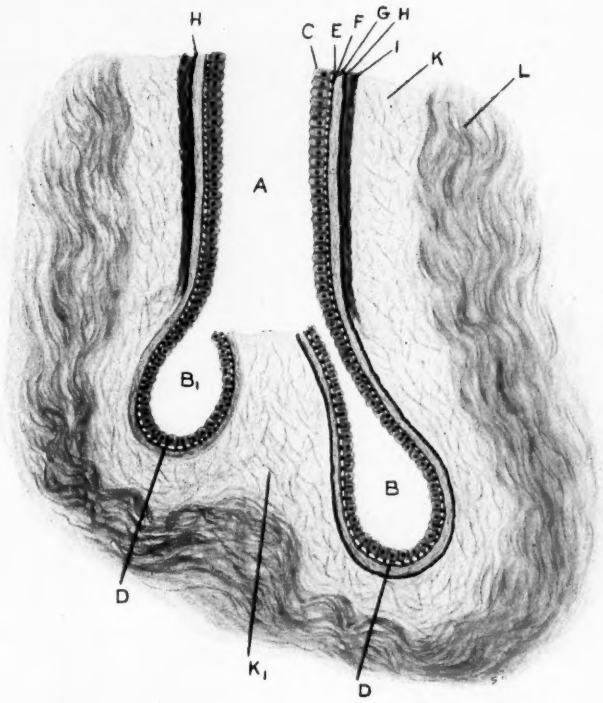

FIG. 334.—Diagram representing a duct and its acini. (A) The interior of the duct. (B) and (B_1) The interiors of two acini. (C) Epithelium. (E) Small crosses representing the single layer of unstriated muscle fibres. (F) Basement membrane. (G) The delicate fibrous tissue to which great attention is drawn in this article. (H) Represents the elastica, to which great attention is drawn in this article. (I) The unstriated muscle tissue of the duct wall. (K) The fibrous tissue outside the duct wall. (K_1) The intralobular fibrous tissue outside the acini; K_1 is directly continuous with K. To K and K_1 great attention is drawn in this article. (L) Represents the general connective tissue of the breast in which all the above tissues are embedded. (D) Indicates the epithelium of the acini, and draws attention to the fact that the tissues of the acini (C, E, F, G) are directly continuous with those in the duct, although the elastica does not surround the acinus B_1.

Class I.—THE HYPERPLASIA INTRA-ELASTICA.

The hyperplasia mainly occurs in the delicate connective tissue which lies immediately internal to the elastica in the ducts and acini (*Figs.* 334, 335, 336, and 348).

The hyperplasia of the intra-elastica connective tissue occurs in breasts over the age

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* On reference being made to " A Further Contribution to the Study of Cysts and Papillomata of the Breast," BRITISH JOURNAL OF SURGERY, 1921, Vol. IX, No. 34, it will be seen that I divide papillomata into uniradicular and multiradicular varieties, the former a rare condition, the latter a common one which bears a very close relationship to cancer. The former arises from one stalk of fibrous tissue ; the latter from many stalks composed of fibrous tissue which is either extra-elastica or intra-elastica in origin, and from stalks composed only of columns of epithelial cells (*see Fig.* 338). All these forms may be seen in the same tumour.

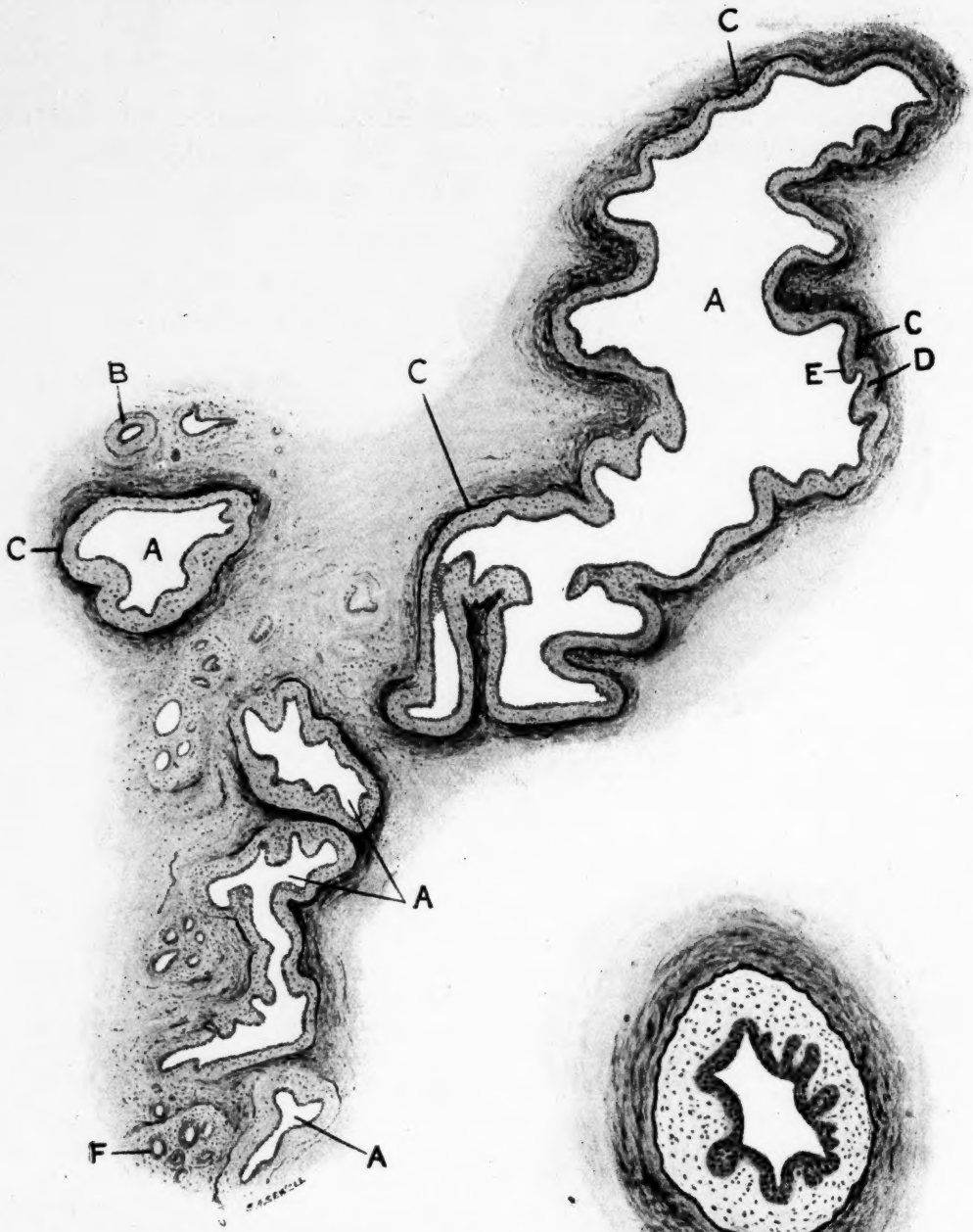

FIG. 335.—From the breast of a married woman, age 43, suffering from cysts, one of which contained a malignant multiradicular duct papilloma. This figure shows hyperplasia of the intra-elastica fibrous tissue before it has undergone degeneration, consisting of scattered bipolar and stellate cells loosely connected together by delicate fibrous tissue. (A) Ducts. (B) Acini. (C) Elastica. (D) Hyperplasia of the intra-elastica fibrous tissue. (E) The epithelium, muscle layer, and basement membrane have been pushed inwards at D. (F) Acini that are not surrounded by elastica. There is no sign of inflammation in the acini and ducts affected.

FIG. 336.—Transverse section of a terminal duct from another part of same breast as *Fig.* 335, in which the intra-elastica hyperplasia of fibrous tissue is seen in a more cellular and earlier state. (a) Elastica. (b) Intra-elastica fibrous tissue. There is no sign of inflammation in this duct.

of 30 years. It manifests itself in two ways: (1) It may be *diffused*, or (2) It may be *localized* as a tumour in a duct or in acini respectively.

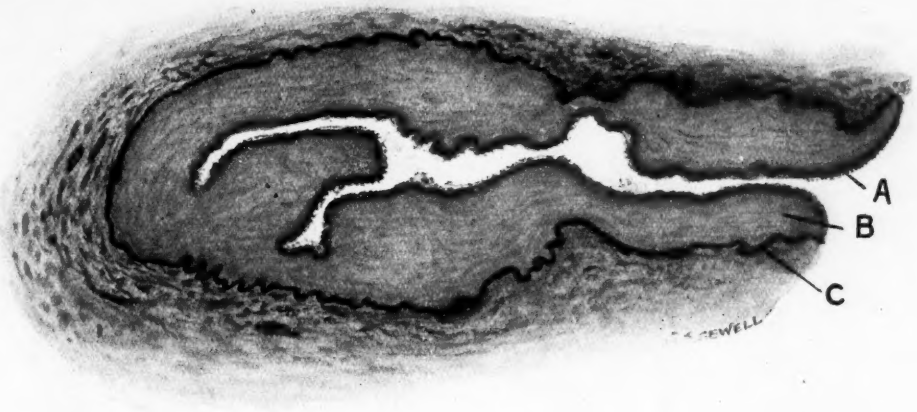

FIG. 337.—From the breast of a married woman, age 38, in which grew the intra-elastica acinous fibro-adenomata referred to in *Fig.* 350.

The figure is a reproduction of a duct. (A) Indicates the epithelial cells, which have in part undergone a desquamative hyperplasia, and in part some of the cells have become squamous in type. (B) The intra-elastica fibrous tissue which has undergone complete degeneration after hyperplasia. (C) The elastica. Serial sections show that this section is made at the centre of a longitudinal duct, and that it is not cut obliquely.

1. In *the diffused condition* the hyperplasia affects the whole length of a duct and all or some of the acini from which it leads, or many ducts and their attached acini.

The hyperplasia of this fibrous tissue can be observed best before degeneration has occurred (*Figs.* 335, 336, and 353). The hyperplasia consists of somewhat large, scattered cells, some of which are bipolar and others stellate, loosely bound together by delicate fibrous tissue. In these figures the complete absence of all inflammation inside or outside the glandular elements should be noted. Degeneration, which as a rule occurs after hyperplasia, may be very widespread (*Fig.* 337, B).

A few small isolated collections of inflammatory cells are sometimes seen in this degenerated intra-elastica tissue, but only rarely; hence they appear to me to be of secondary importance. This diffused type rarely occurs in breasts in which no other lesion can be discovered. The hyperplasia may be found in some breasts in which carcinoma and multiradicular papillomata respectively or combined are present: but it is of importance to note that it is often absent in breasts where carcinoma and multiradicular papillomata, respectively or combined, occur. It can be seen complicating some forms of multiradicular papillomata (*Fig.* 338) and some forms of fibro-adenomata, which belong to the hyperplasia extra-elastica class (*Class III, Fig.* 364), where

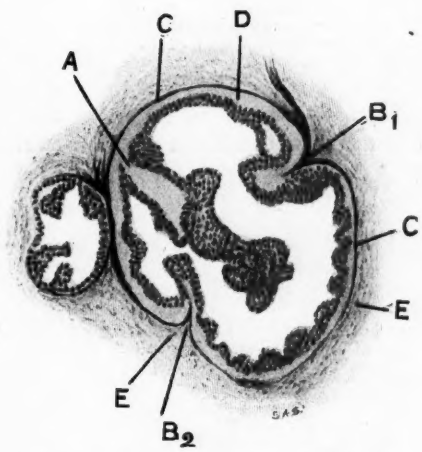

FIG. 338.—From the breast of a single woman, age 35, suffering from duct cancer. Transverse section of a duct which was situated on the internal edge of the breast. The tumour which drew attention to her condition was situated immediately opposite, in the external edge. (C) The elastica. The intra-elastica (D) has undergone degeneration after hyperplasia. (A) A papilloma, the central stalk of which consists only of intra-elastica fibrous tissue hyperplasia. This papilloma is covered by epithelium undergoing very suspicious activity. B_1 and B_2 are papillomata, the central stalks of which contain a slight amount of elastica and extra-elastica fibrous tissue (E). They belong to the hyperplasia extra-elastica type, but they also show marked intra-elastica hyperplasia of fibrous tissue.

it is so marked in amount that it forms the main bulk of the tumour. There are two points of interest in the diffused form of hyperplasia intra-elastica. The first is the recognition of its occurrence in a much greater degree than is supposed, and the second is the fact that localized tumours may arise from it.

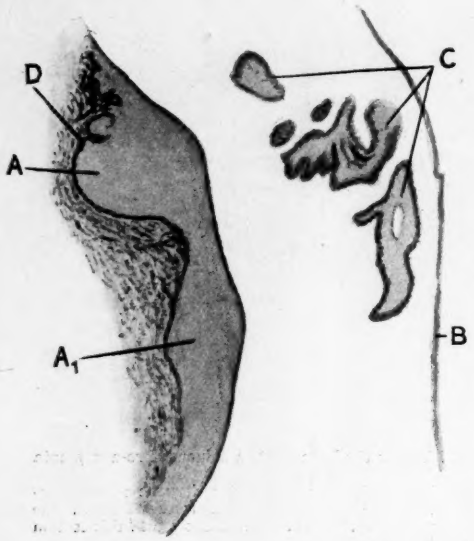

FIG. 339.—From the breast of a single woman, age 31, suffering from discharge of blood from nipple. Longitudinal section of an ampulla of duct. (D) The elastica. A and A_1 represent respectively two oval-shaped masses of degenerated intra-elastica hyperplasia which bulge into the lumen of the ampulla. The epithelial, muscular, and basement membrane layers have been pushed inwards. (C) Parts of a multiradicular papilloma which also grew in this ampulla. (B) The opposite wall of the ampulla represented diagrammatically.

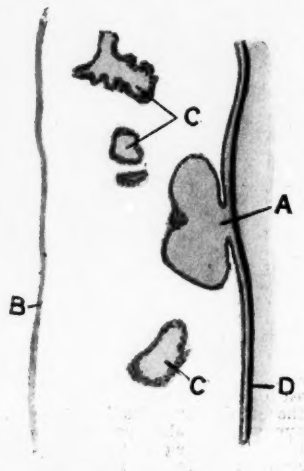

FIG. 340.—From the breast of a woman, age 35, suffering from a discharge of blood from the nipple. Longitudinal section of ampulla of duct. (D) The elastica. (A) A definite pedunculated papilloma growing from the intra-elastica fibrous tissue. (C) Parts of a multiradicular papilloma which grew in this ampulla. There can be seen in other sections of this extra-elastica multiradicular papilloma marked hyperplasia of its intra-elastica fibrous tissue. (B) The opposite wall of the ampulla represented diagrammatically.

2. *The localized condition* is a tumour formation which is usually superimposed on the diffused condition above described. Like the diffused form, it is usually seen in the breasts of women over 30 years of age. The tumour occurs (*a*) in ducts, and (*b*) in acini.

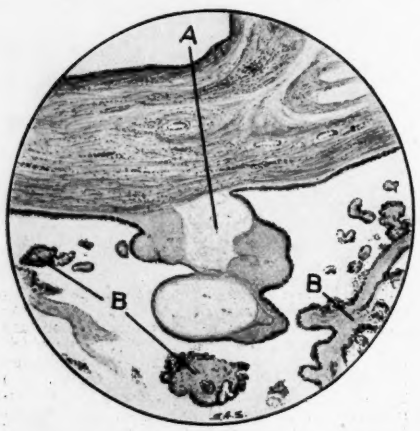

FIG. 341.—A papilloma similar to A in *Fig.* 340, and taken from the same ampulla. (B) Parts of the same multiradicular papilloma seen at C in *Fig.* 340.

a. In ducts.—The simplest tumour formation is shown in *Fig.* 339, in which two oval swellings inside the elastica are placed longitudinally, and bulge into the lumen of the duct. The tissue composing them has degenerated, and has pushed up the epithelial cells together with the subjacent muscle layer, both of which cover their surfaces and appear to be normal. It is important to observe that the duct is also full of multiradicular papillomata. *Figs.* 338, 340, and 341 show more complicated tumours in the definite formation of papillomata. These papillomata grew from the intra-elastica fibrous tissue of separate ducts in separate breasts. Many of the usual type of multiradicular papillomata of the duct exhibit intra-elastica hyperplasia as a complication. By the "usual type of multiradicular papillomata" I mean those which possess a connective-tissue stalk composed of extra- and intra-elastica connective tissues (*Figs.* 338, B_1 and B_2).

From the intra-elastica fibrous tissue may develop large intracanalicular fibro-adenomata. The tumour from which I describe this type of fibro-adenoma grew in the breast of a woman of 51 years (*Fig.* 342). It is a true intracanalicular tumour beginning and spreading in a duct (*Fig.* 342, A, and compare with *Figs.* 335 and 337). This tumour is remarkable in many respects. It clearly began in the form of sessile growths springing from the intra-elastica connective tissue in the walls of a duct. The surfaces of the

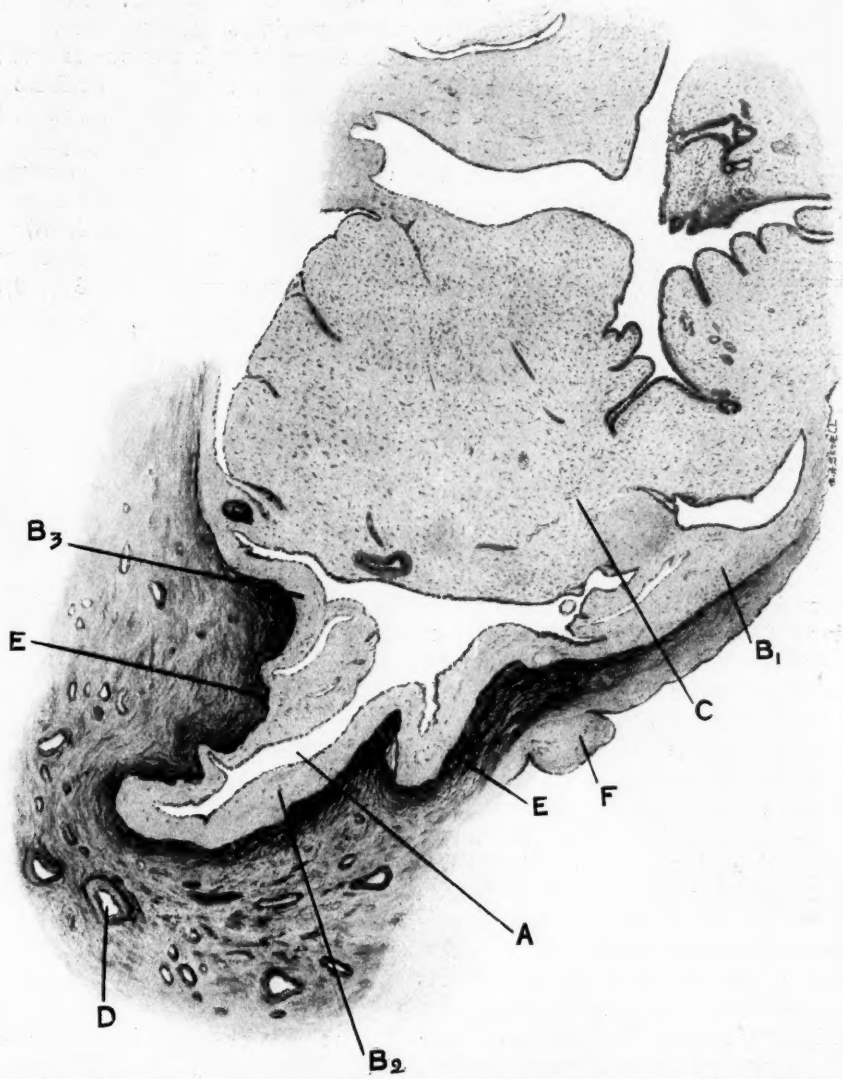

FIG. 342.—An intracanalicular fibro-adenoma removed from the breast of a woman, age 51. (A) Lumen of duct. Compare with ducts in *Figs.* 335 and 337. (B_1, B_2, B_3) Intra-elastica fibrous tissue which has undergone hyperplasia and from which the intracanalicular tumour (C) is growing. (E) The elastica. (D) A small duct cut transversely in which degeneration has occurred in the hyperplasia of the intra-elastica fibrous tissue. (F) Part of the convoluted duct A, undergoing the same changes as A. There is no new formation of elastica; compare with *Fig.* 344, in which it is too extensively represented.

growths are covered by the epithelial and muscle layers. At first the growths are mainly sessile, the margins showing a tendency to pedunculation. The growths may be comparatively far apart and separated, or so close together that their epithelial surfaces are in contact. Some of them become pedunculated with increase in size. The connective

tissue which forms the main bulk of the tumour is chiefly composed of bipolar and stellate cells which are loosely bound together by œdematous and delicate fibrous tissues. In other parts, denser, more fibrous spindle cells are present. A curious feature in the connective tissue of this and allied intracanalicular tumours is the presence of scattered bundles of unstriated muscle fibres; they are not distributed universally over the tumour, but make their appearance only occasionally. I was so much astonished at their presence that I asked Dr. J. A. Murray whether he agreed with me. He said he was quite satisfied they were unstriated muscle fibres.

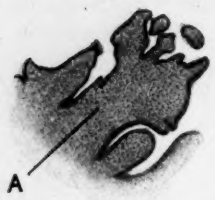

FIG. 343.—Papillomata growing from the surface of the tumour C, *Fig.* 342. From the surface of the papilloma (A), smaller and more pedunculated papillomata are growing. There is no development of elastica at this part of the tumour.

Hyperplasia of epithelium in this tumour is manifested in many remarkable ways. First, from the surfaces of these growths more or less pedunculated papillomata arise, from which develop smaller and more pedunculated papillomata (*Fig.* 343). Further, the epithelial surface is the starting-point of other remarkable changes. Thus, it may dip down into the fibrous tissue and form depressions (*Fig.* 344, A); perfectly normal acini are also developed connected with stunted ducts which open on the surface of the tumour (*Fig.* 344, B). Lastly, comparatively long ducts may dip down from the surface of the tissue and end in a perfectly formed lobule of acini (*Fig.* 344, C). These are new ducts and acini similar to those seen in the healthy, vigorous breasts of young women. These newly-formed ducts always appear as terminal, and are never surrounded by connective-tissue coats (*Fig.* 344). Their points of origin are most irregular and haphazard in their arrangement. The newly-formed ducts and acini in this tumour are derived from duct epithelium, a fact that would seem extraordinary if it were not remembered that all the breast epithelium arises from epiblast.

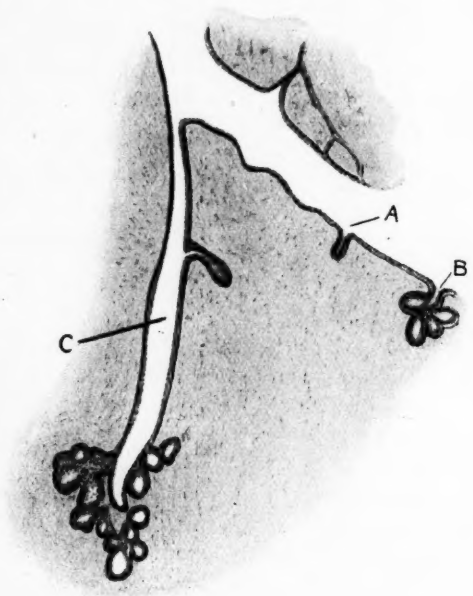

FIG. 344.—Downgrowths of surface epithelium from tumour C, *Fig.* 342. (A) A depression only. (B) Stunted duct opening into acini. (C) A long duct opening into a definite lobule of acini. Elastica of new formation has developed in this part of the tumour. The elastica is drawn too diagrammatically, and is not so continuous nor so marked as shown here. Its occurrence in the tumour is exceptional. (*See Fig.* 342, C.) It will be noted that the ducts have no connective-tissue walls other than the connective tissue of the tumour. The acini are of the vigorous appearance seen in a young breast. The bundles of unstriated muscles scattered among the connective tissue of the tumour, and described in the text, are not shown.

Another curious feature of these newly-formed epithelial structures is that the elastica may here and there develop in its normal relation to them (*Fig.* 344), and often tracts of unstriped muscle are visible between the elastica and the epithelial cells; in some places even intra-elastica hyperplasia can be definitely observed. Very few blood-vessels can be seen. It is difficult to understand how such large tumours can be nourished by means of so small a blood-supply. As long as the growths are within the ducts they are encysted tumours. The pressure produced by the expansion of these tumours in the process of growth destroys the duct wall. The result of this is that the connective-tissue elements of the tumours come in direct contact with the general supporting fibrous tissue, which is so pressed upon that it appears as a definite cyst wall, for which it might easily be mistaken upon casual examination. Careful observation,

however, through serial sections, demonstrates that the compressed fibrous and even elastic tissues are continuous with definite strands which are lost in the structure of the

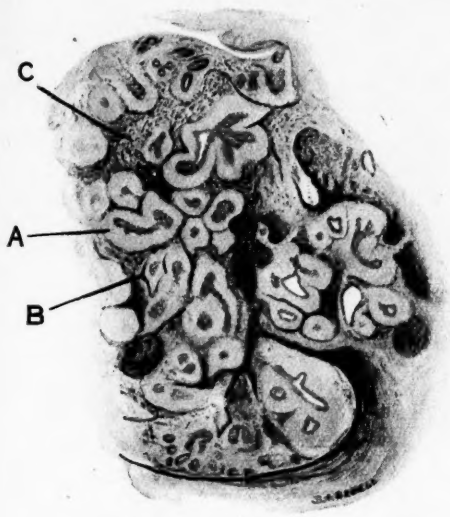

FIG. 345.—From the breast of a married woman, age 43, suffering from cancer. An isolated collection of acini in which the hyperplasia intra-elastica has undergone degeneration. This is a small fibro-adenoma. (A) Intra-elastica hyperplasia of fibrous tissue in acini, some of which are surrounded by elastica (B). The intralobular connective tissue is seen at C. Some acini have no elastica around them.

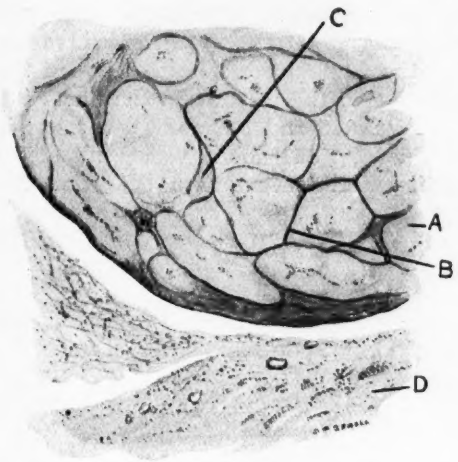

FIG. 346.—From the breast of a married woman, age 45, suffering from cancer. A collection of acini in the margin of a circular fibro-adenoma, the whole of which is composed of the same tissue changes as here shown. In a more advanced degree it shows the same structure as seen in *Fig.* 345. (A) The acini containing the hyperplasia intra-elastica. (B) The elastica. (C) The remains of the intralobular connective tissue. (D) Surrounding tissue.

breast and have nothing to do with duct walls. This remarkable tumour contains bundles of unstriated muscle fibres, fibrous tissue, and epithelial elements ; it also shows papillomata formed upon papillomata, and newly-developed vigorous young glandular tissue, although it grew in the breast of a woman 51 years of age.

In *Fig.* 349, again, is seen an intracanalicular fibro-adenoma of a duct. The tumour resembles in composition that of *Fig.* 342, C, and arises from the intra-elastica tissue.

I believe that many intracanalicular fibro-adenomata belong to this class, and that the extension of their growth has obliterated all trace of their origin.

Lastly, in *Fig.* 353, primary carcinoma in a duct can be seen growing upon the hyperplasia intra-elastica, which is being invaded by the malignant disease at C. I regard the invasion of the structures at C by the epithelial tumour as the earliest indication of invasion by epithelial cells I have seen in a breast, and as a sign that should be sought for in epithelial hyperplasia that is contained within duct structures.

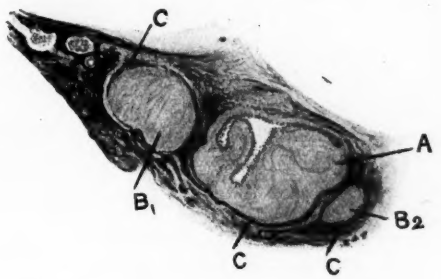

FIG. 347.—From the breast of a married woman, age 41, suffering from many cysts in the breast, one of which contained a multiradicular papilloma. The reproduction shows part of another tumour about the size of a walnut. The tumour was composed of exactly the same type of thing seen in *Figs.* 345 and 346. The three acini (A, B_1, and B_2) show the following points of interest. In A the hyperplasia intra-elastica can be seen to be forming an intra-acinous tumour. B_1 and B_2 are sections of acini which serial sections show contained the epithelial elements seen in A, and therefore they represent the periphery of the hyperplasia intra-elastica in these acini.

b. In acini.—The intra-elastica connective tissue around the acini may undergo so marked a hyperplasia that a collection of acini thus affected resolves itself into a distinct

isolated tumour and forms a fibro-adenoma (*Figs.* 345, 346, 347, and 348). The intra-elastica fibrous tissue may undergo a regular form of hyperplasia round the whole acinus (*Figs.* 345, 346, and 348), or the hyperplasia may be more marked at one part of the acinus and appear as an intra-acinous growth (*Fig.* 347). In the examination of this tumour formation it must be remembered that where the elastica exists the tumour formation is internal to it. Where the elastica does not exist it should be realized that the tumour formation occurs in the same tissues that are directly continuous with those inside the elastica of the ducts (*Fig.* 348).

Figs. 348 and 349 show two reproductions from a breast kindly sent to me by Dr. Creed: *Fig.* 348 shows very beautifully a duct, A, in which there is hyperplasia intra-elastica, terminating in a collection of acini, B, which form a part of a comparatively

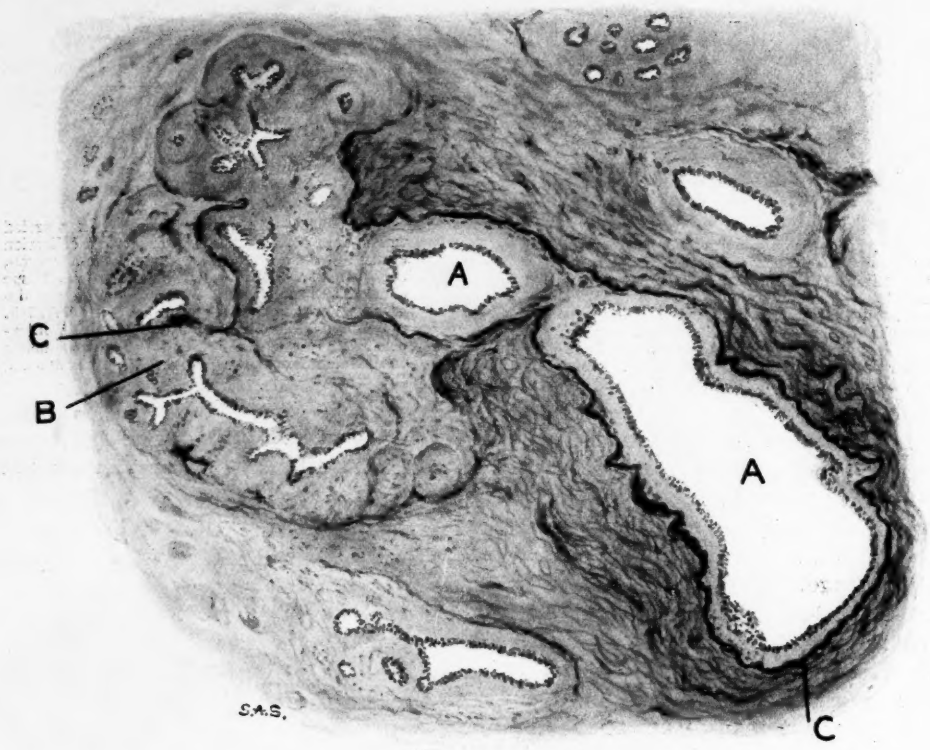

FIG. 348.—From the breast of a woman, age 45, suffering from cancer. (A, A) Parts of a terminal duct cut longitudinally. (B) The acini into which it leads, forming part of a comparatively large fibro-adenoma of the hyperplasia intra-elastica type. (C) Elastica. There is a diffuse hyperplasia intra-elastica in the duct A.

large acinous fibro-adenoma of the hyperplasia intra-elastica type. The elastica is only occasionally present round the acini; where it occurs it is in the normal position. The acinous appearances of this part of the tumour correspond to those one would expect to see if a similar affection arose in the diagrammatic acinus B_1 in *Fig.* 334. There can be no doubt that the hyperplasia intra-elastica of the duct is continuous with that of the acini. A and A, *Fig.* 349, are two parts of the same convoluted duct which have been cut transversely. (Compare with the same occurrence in *Fig.* 342, F and A.) In the two sections of the same duct are seen the beginnings of intracanalicular fibro-adenoma of the hyperplasia intra-elastica type. The pathological anatomy of these growths exactly resembles the intracanalicular tumour in *Fig.* 342, C.

These tumours may appear alone, or they may complicate other forms of fibro-adenomata (*Class III*). In either state they are composed of bipolar or stellate cells, loosely connected by fibrous tissue, or more commonly they may be so degenerated as to exhibit little evidence of previous fibrous structure (*Figs.* 345, 346, 347). I have no doubt their clinical signs are put down to what is called 'localized chronic mastitis', with which it has no relation.

I have no specimen in my possession which shows carcinoma arising in an acinus in which there is hyperplasia intra-elastica. *Fig.* 353 is an example of carcinoma involving a duct thus affected.

Before leaving the subject of intra-elastica hyperplasia of connective tissue, it may be well to recall that Ribbert observed the condition, and stated that he regarded it as of vast importance in the carcinoma process. I have seen this hyperplasia in breasts in which neither malignant nor benign tumours were present, and also in breasts in which

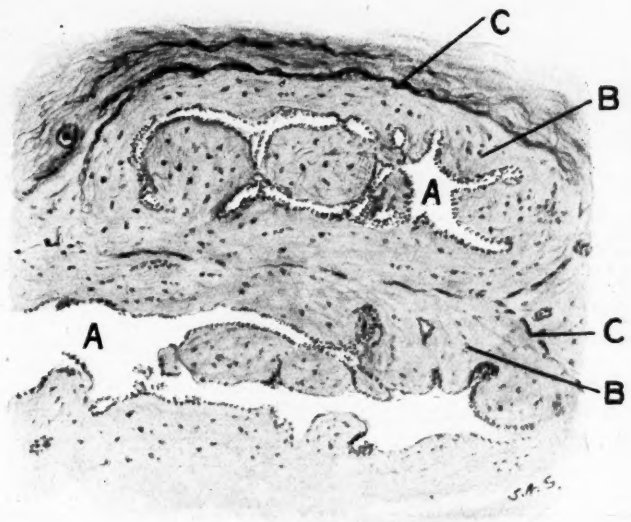

FIG. 349.—From the same breast as in *Fig.* 348. Two parts of a convoluted duct cut transversely (A, A). The duct was situated in close vicinity to the section shown in *Fig.* 348; in it are seen the beginnings of an intracanalicular fibro-adenoma arising from the intra-elastica (B) of both sections of the tube. The growth exactly resembles the structure of the tumour in *Fig.* 342, C. (C) Elastica.

benign and malignant tumours were present in the same glands; yet I can observe nothing to contra-indicate the assumption that epithelial activity is the primary change in carcinoma. Victor Bonney, in his article on "The Connective Tissues in Carcinoma and in certain Inflammatory States that Precede its Origin", published in the *Archives of the Middlesex Hospital*, xiii, p. 24, shows two illustrations, Nos. 21 and 22. In No. 21 he depicts a duct in which the connective tissue between the epithelium and the elastica has undergone hyperplasia in a breast which he describes as suffering from 'chronic mastitis', and in No. 22 he depicts a duct in the same state, but in part of a breast invaded by carcinoma. I should include the specimens from which these figures are taken under my *Class II*, the hyperplasia elastica, which occurs very frequently where there is no hyperplasia intra-elastica, and I do not believe that their relations to an 'inflammatory state' are fundamental. In the early state of hyperplasia intra-elastica inflammatory signs are absent.

Class II.—THE HYPERPLASIA ELASTICA TYPE.

In this class, in which there is a combined hyperplasia of the elastica and the fibrous tissue with which it is intermingled (*Figs.* 350 and 351), the limitation of the growth is

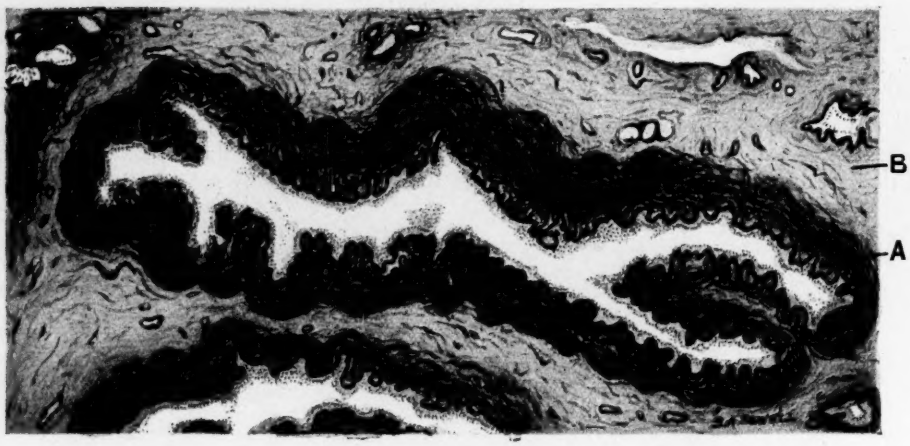

FIG. 350.—From a breast of a married woman, age 38. The gland was removed for a painful nodular condition over the whole breast. Most of the nodules were clearly and clinically demonstrated to be irregularities of enlarged and tortuous ducts. It was diagnosed as being a 'proemial breast' (*see* text and footnote). The drawing shows a longitudinal section of a duct. The elastica (A) has undergone enormous hyperplasia, the boundary of which is limited at the margin of the fibrous tissue which closely invests the duct (*Fig.* 351, A). (B) The connective tissue which supports the general structure of the breast. A section stained for fibrous tissue is seen at A in *Fig.* 351.

In *Figs.* 350 and 351 desquamative hyperplasia of epithelium is seen in the duct, and occasionally in the duct in *Fig.* 350 slight hyperplasic intra-elastica can be seen.

fixed at the margin of the fibrous tissue that immediately surrounds the ducts and acini (*Fig.* 334, K and K_1, and *Fig.* 351, A). Most frequently the process is widely distributed,

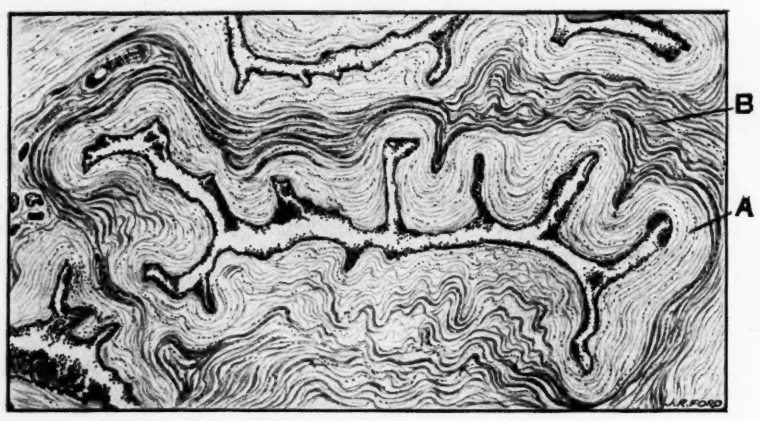

FIG. 351.—The same duct as in *Fig.* 350, in the next section of the series. The elastica has not been stained. This section was stained to show the increase of fibrous tissue (A), which is intermingled with the hyperplasia elastica in *Fig.* 350, A. (B) Shows the fibrous tissue which is supporting the breast tissues in the general way. The delimitation of the fibrous tissue A from the fibrous tissue B is most marked all round the duct. The Wassermann reaction was negative.

and affects most of the ducts and acini in a breast. It is accompanied by desquamative hyperplasia of epithelium. Specimens in which I have observed it have been breasts in

which carcinoma existed, and those which I have ventured to term 'proemial breasts'* (*Fig.* 352); also in breasts which suffered from Paget's disease of the nipple, where ducts and acini were full of carcinoma (*Fig.* 353). I am so impressed with these facts that I should be most careful to examine the entire breast in which there was enormous hyper-

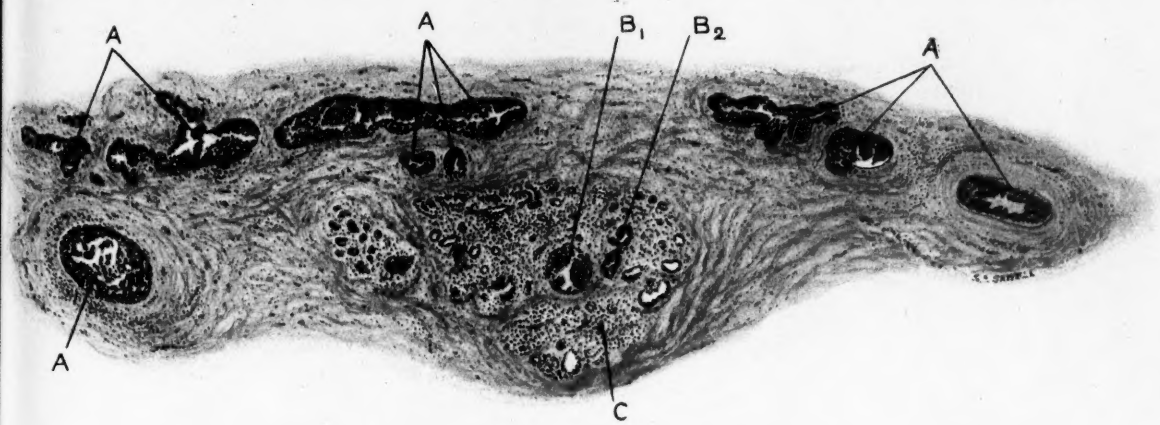

FIG. 352.—A reproduction of another duct and lobule from the same breast as in *Figs.* 350 and 351. The elastica is not stained in this section. Serial sections show that the tissues marked A all belong to one duct, a branch of which ends at B_1 and B_2 in the lobule C. The hyperplasia of the epithelium seen in this duct is not desquamative in type. In my opinion it is carcinoma, although the epithelial cells remain within all the walls of the duct. I consider this type of epithelial hyperplasia renders justifiable the term 'proemial' that I applied to this breast.

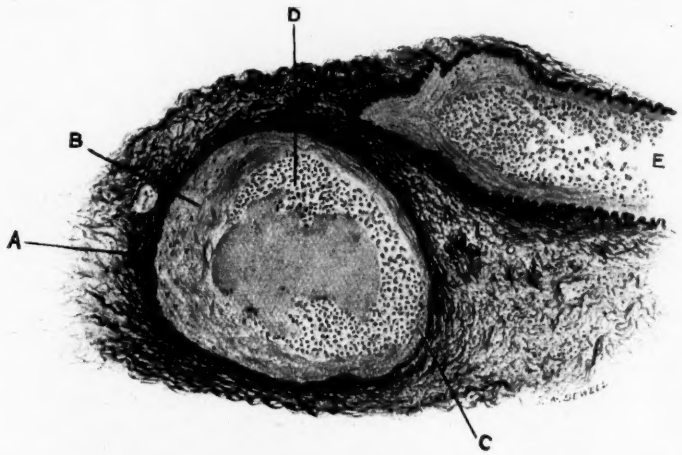

FIG. 353.—From the breast of a woman suffering from Paget's disease of the nipple. One or two ducts and their acini were filled with carcinoma which had spread into the lymphatic vessels. All the ducts and most of the acini showed the same enormous hyperplasia elastica as seen in *Fig.* 350. The figure is a transverse section of a duct. (A) The hyperplasia elastica (B) Hyperplasia intra-elastica, which is being invaded at C by the carcinoma D within the duct. It has not invaded the hyperplasia elastica A. (E) A longitudinal branch of the same duct; it is full of carcinoma which has not invaded the hyperplasia intra-elastica. In serial sections this longitudinal branch terminates in acini, and is not a blood-vessel as its appearance suggests.

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* The term '*proemial breast*' is employed to indicate a condition of the breast which makes it a prelude to later developments of papillomata and of duct and acinous carcinomata. A 'proemial breast' can be recognized clinically by the presence of cysts, and in its earlier stages by the irregularity in the size of a duct or ducts. Ducts can be rendered irregular in size: (1) By a desquamative hyperplasia of epithelium; (2) By a hyperplasia of epithelium which is not papillomatous or desquamative in type, but which in my opinion is malignant (*Figs.* 352, 353); (3) By a combined hyperplasia of elastica and fibrous tissue intermingled with it (*Figs.* 350, 351); (4) By a hyperplasia of the intra-elastica fibrous tissue (*Figs.* 335, 336, 337); and (5) By papillomata which may be benign or malignant. These five forms of duct irregularity are often indistinguishable clinically from each other, and all are now being diagnosed as 'chronic mastitis'. For further reference see "Cancer of the Breast", *Brit. Med. Jour.*, 1922, June 3.

plasia of the elastica before I passed the gland as free from carcinoma. On the other hand, there can be no doubt that most breasts which contain carcinoma do not show such enormous hyperplasia of the elastica, and the same may be said of breasts containing multiradicular papillomata. It occurs both in syphilitic patients and in patients in whom no evidence of syphilis can be obtained. In a syphilitic woman who had carcinoma in both breasts, this enormous hyperplasia of the elastica was present in both glands (*Fig.* 354).

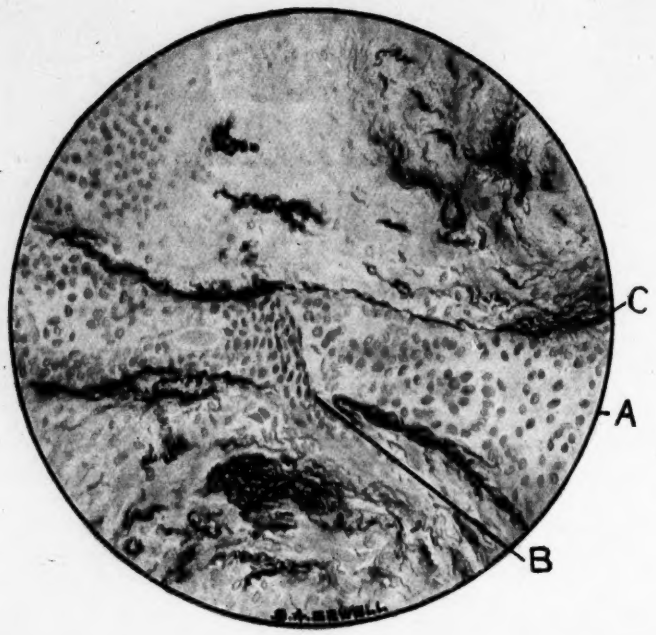


FIG. 354.—From the breast of a woman, age 42, suffering from carcinoma of both breasts and a positive Wassermann reaction. The hyperplasia elastica was as great as that seen in *Fig.* 350 in the ducts and acini of both breasts. The figure is a reproduction of a small terminal and longitudinal duct. It contains carcinoma (A), which has invaded the hyperplasia elastica at B. (C) Hyperplasia elastica, which was very irregular in distribution in this part of the terminal duct. There was no hyperplasia intra-elastica in this breast, nor in the opposite breast.

The condition may be uncomplicated by the diffused hyperplasia of intra-elastica fibrous tissue, or may be accompanied by it to a varying degree. I can see no reason for not considering this condition a diffused form of fibro-adenoma, although I have never seen it as a localized tumour. The outer coats of the arteries in these breasts show an irregular hyperplasia of their elastic fibres.

In *Fig.* 354 is seen an early cancer invading the elastica in a condition of this kind. It has invaded the elastica before it has penetrated the duct wall ; in a section not stained to show elastic tissue this observation could not have been made.

Class III.—THE HYPERPLASIA EXTRA-ELASTICA.

This class concerns the fibrous tissue immediately outside the ducts, and the intra-lobular connective tissue of the acini (*Fig.* 334, K_1 and K), and includes mainly well-known varieties of fibro-adenoma of the breast.

Extra-elastica fibro-adenomata are formed in the terminal segments of a breast, and affect ducts and acini. I divide this class into: (1) Pericanalicular and peri-acinous, which are (*a*) localized fibro-adenomata (*Figs.* 355, 356, 359–367), or (*b*) diffused to form a general adenomatous state of the breast ; and (2) Intracanalicular (*Figs.* 357 A, 359 C, 360 A, 364). Except in the type of the diffused fibro-adenomatosis, which occurs after 30 years, all forms may grow in young and old breasts.

1. *Peri-acinous and Pericanalicular.—a. Localized Form.* The terminal ducts and acini are, as a rule, involved in this formation. The epithelial changes show themselves by a desquamative hyperplasia within the lumina, and spread in circumferential and longitudinal dimensions as the tumour grows; and, although the hyperplasia of the fibrous

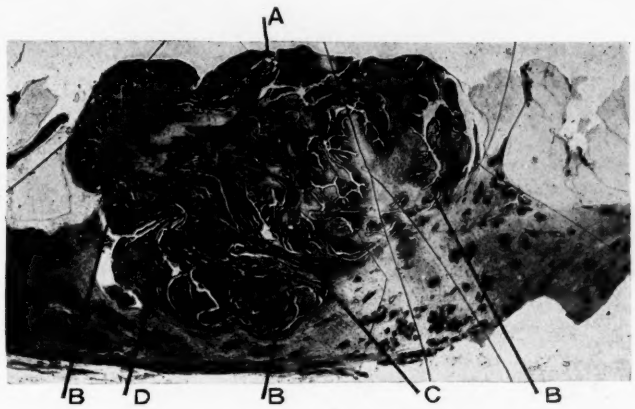


FIG. 355.—Segment of breast containing a fibro-adenoma. Hyperplasia extra-elastica, from female, age 23. The tumour is mainly a peri-acinous and pericanalicular fibro-adenoma in which there is an intracanalicular growth, the precise origin of which I cannot discover. (A) Upper edge of the tumour which impinges on fat. Here it has no capsule. (B) Lower edge of tumour, round which the surrounding breast tissue appears as a pseudo-capsule. (C) Portion from which *Fig.* 356 is reproduced under higher power. (D) Portion from which *Fig.* 357 is reproduced under higher power.

tissue manifests itself primarily in the particular parts to which I have referred (viz., in the fibrous tissue that closely invests the outside of ducts and individual acini), as the tumour increases in age and size the fibrous tissue supporting adipose tissue may become affected, and the tumour may thus include fat within its boundaries (*Fig.* 361). Mainly,

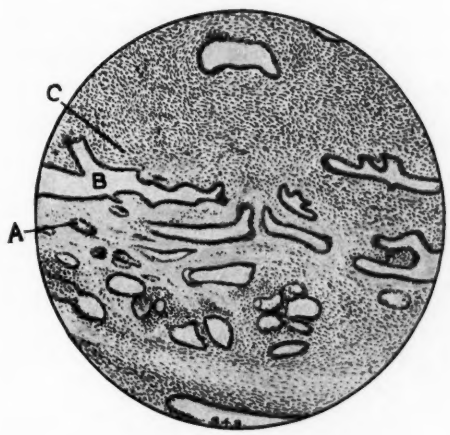


FIG. 356.—From C in *Fig.* 355. (A) A less cellular part of the specimen, and continuous with the normal structure of the breast at C in *Fig.* 355. (B) A duct, the upper part of which is involved in the hyperplasia extra-elastica of the peri-acinous and pericanalicular tumour (C). The lower part is still in the unaffected region (A). Elsewhere in the section the peri-acinous and pericanalicular change is occurring.

however, the coarse fibrous tissue which supports the breast is pressed upon by the growth, and thereby made to appear as a capsule; but where the tumour impinges on fat there is no capsule (*Fig.* 355, A). It is important to observe that the tumour increases in size in two ways: first, by hyperplasia of the epithelial and fibrous tissue in the original tumour formation, and secondly, by an exactly similar affection occurring in fresh areas of the breast. The newly-affected areas may be in juxtaposition to the original tumour (*Fig.* 366), or they may be separated from it by comparatively normal breast tissue (*Fig.* 365); then serial sections show that the fresh area may be quite separate

from the original tumour although it may be forming in part of the same segment of the breast. The appearances in *Figs.* 355–363 and 367 bear testimony to this fact. The importance of this observation lies in the inevitable conclusion that this so-called fibro-adenoma is really a process affecting consecutive parts in a localized area of a gland that has been normal. It is wonderful to notice the sharp area of distinction that exists

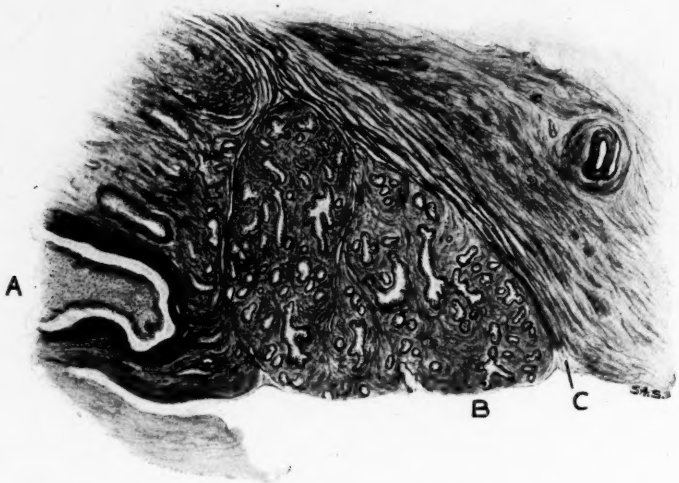


FIG. 357.—Edge of tumour at D in *Fig.* 355. An intracanalicular complication to the tumour has occurred at A, the origin of which I cannot trace. At B a peri-acinous and pericanalicular hyperplasia of the extra-elastica fibrous tissue is occurring round glandular elements. The supporting fibrous tissue of the unaffected part of the breast is being pressed upon by the growth at C. The area B is part of the tumour.

between the hyperplasia of the fibrous tissue immediately surrounding ducts and acini (*Fig.* 359, B_1, B_2, B_3, B_4) and that which forms the coarser supporting elements of the breast. In this type there does not seem to be any marked increase in the actual number of ducts and acini. These structures appear to be widely separated from one another, and do not show or suggest a multiplication of their members. This tumour forms the peri-acinous and pericanalicular fibro-adenoma.

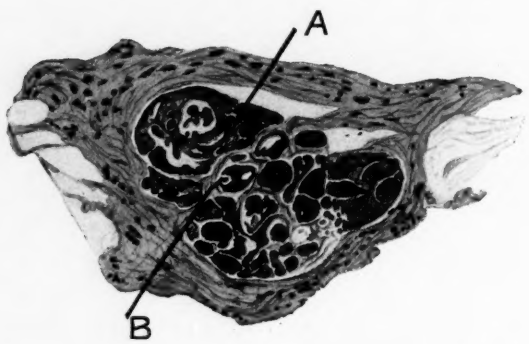


FIG. 358.—A segment of a breast from a woman, age 25. It contains a fibro-adenoma mainly composed of intracanalicular growth A, the origin of which I cannot trace. (B) The part which is peri-acinous and pericanalicular, and the tumour arises from the extra-elastica fibrous tissue around ducts and the intralobular tissue round the acini.

b. A diffused form of extra-elastica hyperplasia of fibrous tissue may occur in a peri-canalicular and peri-acinous form, and may affect practically the whole of a breast. The condition resembles microscopically the appearance seen in the localized condition of peri-acinous and pericanalicular fibro-adenomata. Among this diffused condition can be seen an occasional small isolated tumour which exactly resembles a peri-acinous and pericanalicular fibro-adenoma (*Fig.* 367). The diffused fibro-adenomatous condition of

breasts occurs after the age of 30, and some observers are inclined to doubt whether the small isolated tumours discovered in them are of precisely the same nature as those which

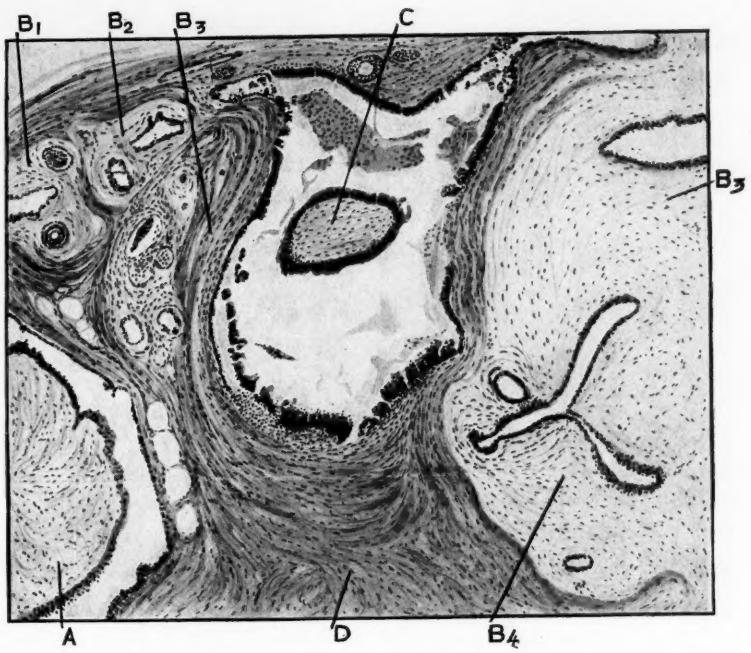
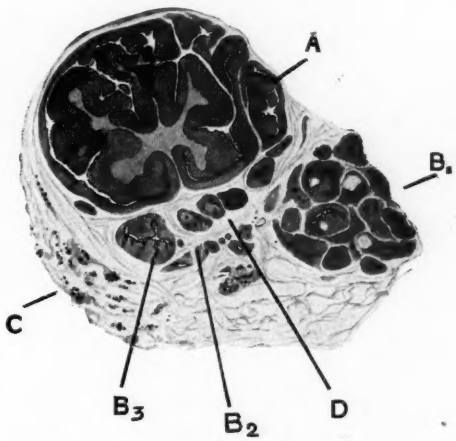


FIG. 359.—A reproduction under higher power of the part marked B in *Fig.* 358. B_1, B_2, B_3, and B_4 show the peri-acinous and pericanalicular hyperplasia of the extra-elastica tissue. (D) The unaffected supporting connective tissue of the breast. A and C are intracanalicular tumours which complicate the tumour.

occur in younger people. I can only say there is no difference between them in their growth and microscopical appearances. I believe that the diffused pericanalicular and peri-acinous condition I have described is a primary condition, and not merely the result of chronic inflammation.

FIG. 360.—Segment of a breast from a woman aged 42. It contains a large fibro-adenoma that had existed for ten years. The main size of the growth is due to the large intracanalicular growth at A. B_1, B_2, B_3, and all the smaller of the deeply-stained parts, are composed of peri-acinous and pericanalicular growth of the extra-elastica fibrous tissue. There is no capsule to this tumour. (C) Normal breast tissue. At D there is a duct which is reproduced under higher power in *Fig.* 361.



2. *The Intracanalicular* (*Figs.* 357 A, 359 C, 360 A, and 364).—The intracanalicular type is commonly complicated by the peri-acinous and pericanalicular changes similar to those that occur in the localized fibro-adenomata of that type. Both types commonly exist in the same tumour; there are many tumours in which the intracanalicular condition is so

pronounced that it forms the predominating part of the tumour. Sessile or more or less pedunculated growths bulge into the lumina. They are composed of wide central stalks

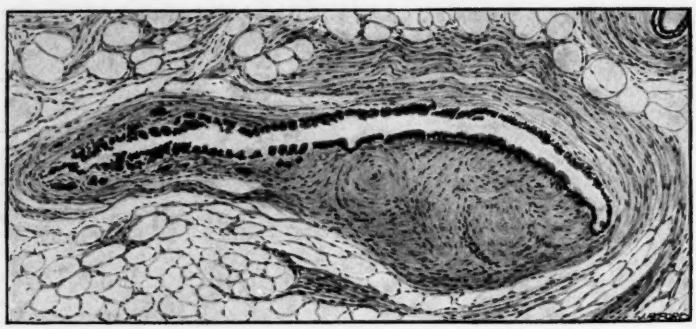


FIG. 361.—Section of duct from D in *Fig.* 360. There is hyperplasia extra-elastica of this duct, and there is marked hyperplasia in the fibrous tissue which is supporting the fat around the duct.

of dense fibrous tissue in the centre of which the elastica is embedded, and it is because the connective tissue outside the elastica grows into the lumina that I place the tumour in the extra-elastica class. This type of tumour is characterized by enormous variations in the amount of intracanalicular growths. There may

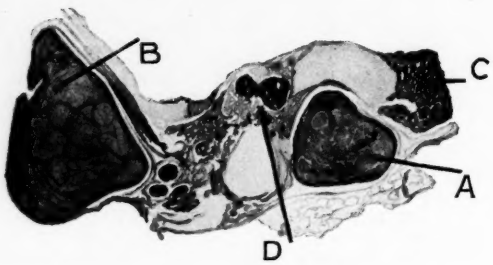


FIG. 362.—Portion of a breast from a woman aged 35. Three separate fibro-adenomata (A, B, C) are seen. They are hyperplasia extra-elastica in type. D marks situation of portion reproduced under higher power in *Fig.* 363, A.

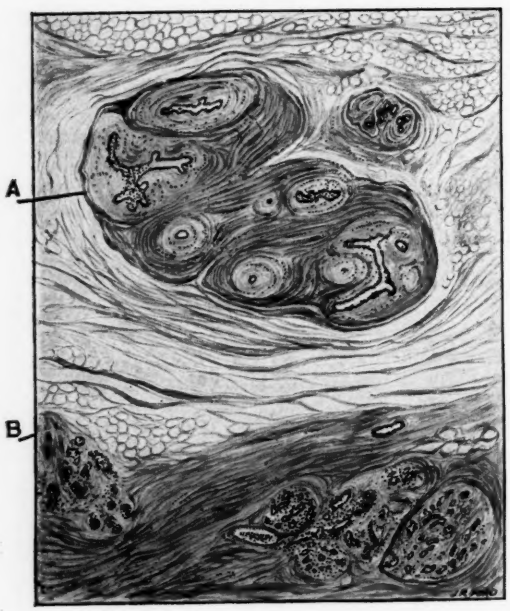


FIG. 363.—Higher power view of *Fig.* 362, D. (A) Peri-acinous and pericanalicular hyperplasia extra-elastica.

be only one or two ingrowths, complicating a peri-acinous and pericanalicular tumour, or the ingrowths may be the main structure of the tumour (*Fig.* 360). From the intracanalicular growths lateral outgrowths may arise (*Fig.* 364). In many of these tumours very marked hyperplasia occurs in the intra-elastica subepithelial tissue (*Fig.* 364). The intra-elastica complication may be so enormous as to form the main bulk of the tumour (*Fig.* 364). There is not the same tendency to form new ducts and acini as can be observed in the *Class I* intracanalicular fibro-adenoma. No doubt some of the intracanalicular tumours which I have been unable to classify may belong to the intra-elastica *Class I* type, all indications of their origin having been lost by the growth and spread of the tumours.

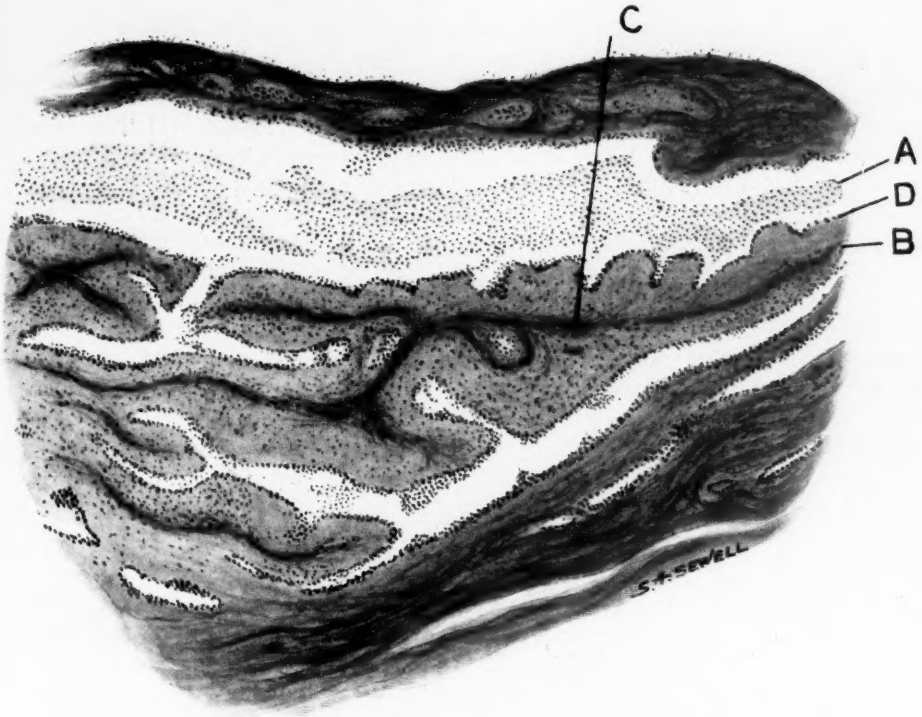


FIG. 364.—Part of the fibro-adenoma A in *Fig.* 362, reproduced under higher power, showing an intracanalicular growth (B), the stalk of which contains elastica (C) in the centre of connective tissue. The tumour belongs to the hyperplasia extra-elastica type. Between the elastica of the stalk and the epithelium (D) hyperplasia intra-elastica has occurred and forms the main bulk of this tumour. (A) A desquamative hyperplasia of the epithelium. It is difficult to say whether this tumour be duct or acinus. The amount of elastica in the stalks of the ingrowth would suggest a duct structure.

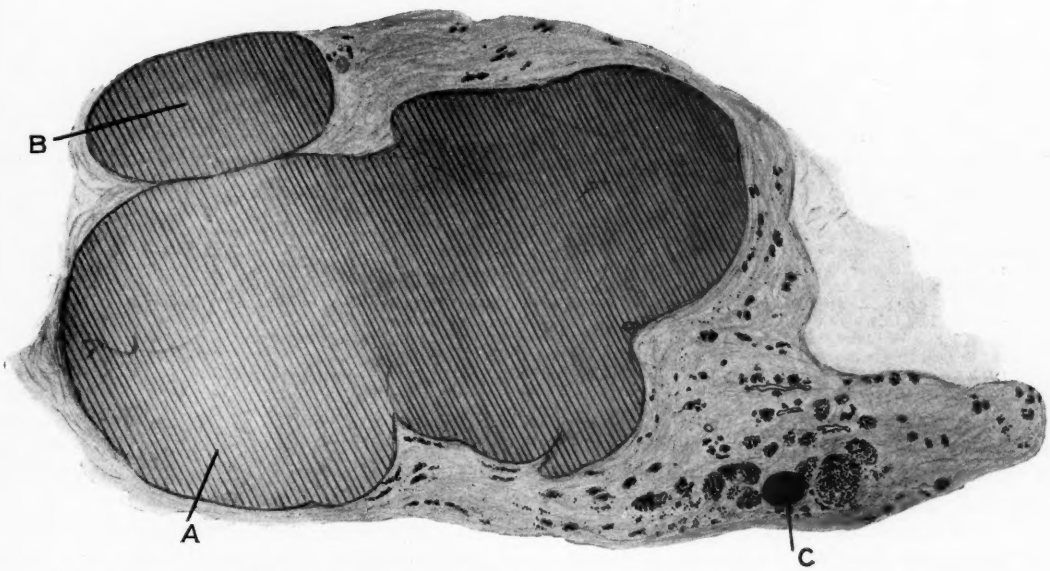


FIG. 365.—Segment of a breast from a woman, age 22, suffering from the fibro-adenoma which is represented in the diagram. The tumour (A and B) belongs to the hyperplasia extra-elastica class. (C) A fresh area of the same process; in its vicinity more lobules are showing the beginning of the same changes in the extra-elastica tissue.

GENERAL OBSERVATIONS.

From the above description of the hyperplasia of epithelial and connective tissues of the breast it will be noticed among other things that a new classification of fibro-adenomata emerges. I divide fibro-adenomata into three classes: (I) The hyperplasia intra-elastica class, which can be subdivided into (1) tumours affecting ducts, (2) tumours affecting acini, and (3) a diffused form affecting ducts and acini; (II) The hyperplasia elastica class, which exists only in the diffused form affecting ducts and acini; and (III) The hyperplasia extra-elastica class, which can be subdivided into (1) pericanalicular and peri-acinous tumours, of which there is (*a*) a localized form and (*b*) a diffused form, and (2) the intracanalicular tumours. It may be interesting to state that I believe it is exceedingly rare for carcinoma to originate in a localized fibro-adenoma. I have only one example of this occurrence. I have another example of a fibro-adenoma being invaded by carcinoma which began elsewhere.

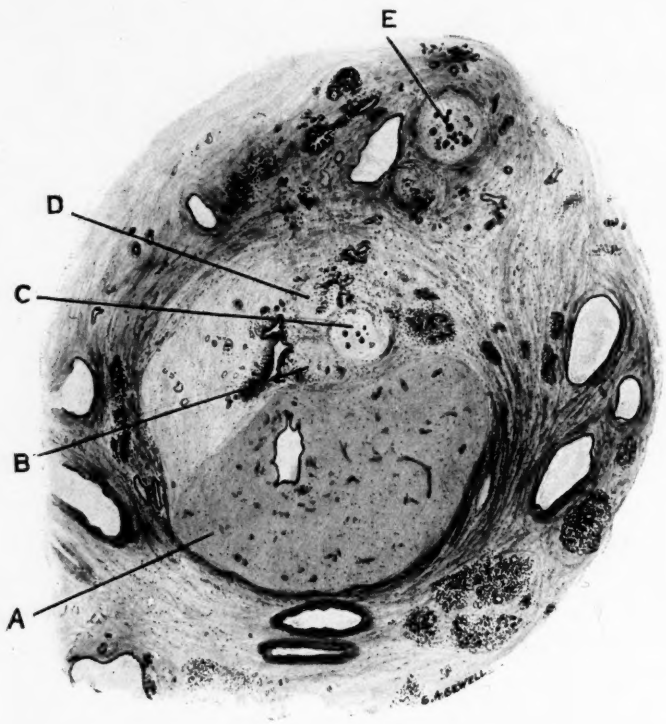


FIG. 366.—From a married woman, age 41, suffering from pain in breast, and as the opposite gland had been removed for cancer she insisted on the removal of its fellow. On microscopical examination of large sections, multi-radicular papillomata were discovered in the ampullæ of two ducts, and the fibro-adenoma which is here reproduced. The fibro-adenoma belongs to the hyperplasia extra-elastica peri-acinous and pericanalicular class. The terminal ducts and acini of this part of the breast are affected. (A) The fibro-adenoma. (B, C, D) Fresh areas undergoing the same process and in juxtaposition to the tumour. (E) A fresh area of the same process at some distance from the tumour.

Compare the appearances of this section with those of *Figs.* 356, 357, 359, 360, 361, 363, 365, and 367, all of which support the statement in the text that a localized peri-acinous and pericanalicular fibro-adenoma is a process that attacks parts of a breast consecutively.

Unsuspected fibro-adenomata, often only just visible to the naked eye when examining a section of a whole breast, occur in 28 per cent of all breasts containing carcinoma; this is probably a low estimate.

It appears to me impossible to attach any special clinical signs to the different types of tumours. The only practical outcome of my observations seems to be that it is safer to take away the segment of the breast in which fibro-adenomata grow. The successful removal of a fibro-adenoma by enucleation is the result of a lucky accident.

Pathological investigation of fibro-adenomata forms practically an untouched subject, and careful work in this direction is essential to isolate so many varieties. It can be inferred correctly that there is no such thing as a fibro-adenoma that does not fall into one of the three groups I have described. It can be inferred also that papillomata and intra-cystic growths of the breast fall naturally into the same classification.

In this paper I have described three distinct classes into which many diverse pathological changes naturally fall. I wish to impress upon the reader the fact that one class may be complicated by the presence of some pathological changes which I have placed in one of the other classes. The occasional overlapping of one class by another occurs

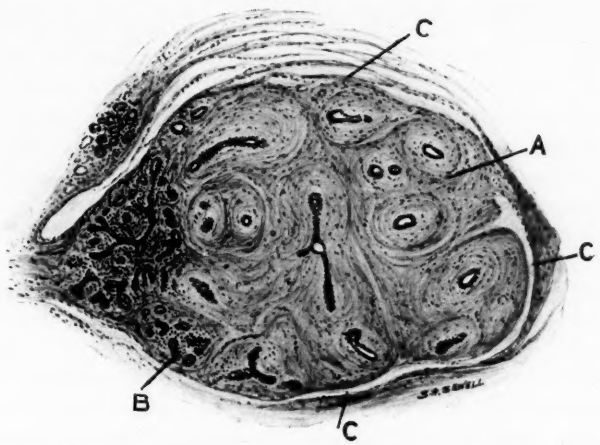


FIG. 367.—From the breast of a woman whose gland was removed because of the possibility of the condition around the nipple being Paget's disease. There was no Paget's disease. The small fibro-adenoma A belongs to the hyperplasia extra-elastica class, and is in a typical manner affecting only the intralobular connective tissue. The supporting connective tissue of the breast at C is not taking active part in the changes. In the lobule B the peri-acinous and pericanalicular changes seen at A are beginning.

so definitely that the trend of my observations is as follows : All the pathological changes to which I have drawn attention (and I include carcinoma) may be phases of a consecutive evolution of disease. I do not wish my classification to incur the penalty of obscuring this probable correlation ; were that to happen it would destroy a notion that pervades the whole of my article.

Lastly, there is another feature in my work I wish the reader to consider. Observers recognize that all epithelium that dips down into deeper structures is not necessarily malignant ; yet they do not seem to me to take into serious consideration the idea that all hyperplasia of epithelium that is confined within the ducts of the breast is not necessarily benign. I am convinced that epithelium thus situated may be malignant. The hyperplasia of epithelium in *Figs.* 353 and 354 shows instances of what I consider to be malignant disease.

ACTINOMYCOSIS OF THE RIGHT ILIAC FOSSA.

By R. ST. LEGER BROCKMAN, Sheffield.

Whilst an unbridled habit of attempting to bring about an alteration in the accepted nomenclature of a disease has nothing to commend it, I must plead a certain justification for the somewhat unusual title of this paper. The time-honoured name of ileocæcal actinomycosis has been discarded here in favour of one which, though admittedly somewhat clumsy, gives a more accurate description of the pathology of this condition. The more familiar name of ileocæcal actinomycosis undoubtedly owes its existence to the apparent but superficial similarity between this disease and that due to an infection by *B. tuberculosis*. In this latter condition the main portion of the disease is confined to the coats of the ileum, cæcum, and appendix. In cases of infection in this region with *Actinomyces bovis*, investigations go to show that the intestine and appendix are themselves free from actual lesions, whilst the muscles and connective tissues of the right iliac fossa are the primary seat of the disease. Therefore, as a result of the conclusions arrived at in this paper, I have attempted to find a title which would convey a more accurate idea of the true etiology of this condition.

Invasion of the right iliac fossa with the organism of actinomycosis is not as rare a condition as some accounts might lead us to believe. A perusal of recent literature tends to give the impression that cases of this infection are becoming more frequent. A truer explanation of the apparent increase lies, I feel sure, in our greater facilities for, and our improvement in, diagnosis. Most observers agree that about 20 per cent of actinomycotic infections in man occur in the intestinal tract. In the main such lesions are located in the region of the appendix and cæcum, whilst a few have been recorded as occurring primarily about the sigmoid or rectum. The stomach and small intestine appear immune or nearly so, since even in the recorded cases there is some doubt whether such lesions were really the primary seat of the disease.

The earliest clinical description of this disease in the right iliac fossa that I can find in the surgical literature of our own land was published in 1892 by Ransom in the *Transactions of the Medico-Chirugical Society*. Like many of the recorded cases of that period, the article affords most delightful reading, being full of accurate clinical observations. This case and the post-mortem descriptions found there are referred to in a later portion of this paper. The disease in this locality, as in other parts of the body, is essentially of a chronic nature. Here, unfortunately, its course is more rapid and more fatal than in other situations in the body.

In most of the earlier cases the observers have pointed out that the disease in the ileocæcal region manifests four stages. The first with varying abdominal symptoms mainly confined to the right iliac fossa ; the second with the presence of a tumour in that locality ; the third with sinus and fistula formation ; and the fourth with processes of repair, or more often with a gradual decline ending in dissolution.

Of recent years a change has taken place in the type of case which is reported. Observers are recording the persistence of a sinus after removal of the vermiform appendix for what was first thought to be a case of ordinary acute or subacute inflammation of that organ. Sooner or later this sinus was found to yield the sulphur granules of actinomycosis. There is, I think, an easy and reliable explanation of this apparent change. We are really dealing with two different stages in the course of identical conditions of disease. It is interesting to read in the records of Ransom's case, of the methods of treatment which were adopted even when the tumour formation was of large extent. It was evidently a

fearful thing in those days to deal surgically with even an inflammatory swelling in such a situation. At the stage of our medical knowledge then reached the disease did not come under the ken of surgeons until it was well advanced and the whole of the ileocæcal region involved. On carefully perusing the early stages of the histories of these cases one is forced to the conclusion that the first symptoms were those of inflammation of the appendix, and in these days the patients would have been operated on much sooner. I feel certain this is exactly what is happening at the present time. Most of the cases recorded in recent literature describe the disease as following the removal of the cæcal appendix. The stages of this condition subsequent to appendicectomy are still identical with those of the earlier cases, which were only first seen when tumour formation was already an accomplished fact. Such evidence is sufficient to suggest that these cases, seen at an earlier stage, should be able to throw some light on the primary pathological lesions of the so-called ileocæcal actinomycosis.

PATHOLOGY.

Formerly, in consequence of the tumour formation and the extent of the disease when first seen, the actual anatomical site of the original focus of infection was the subject of argument and debate. It was difficult, on account of the extensive progress of the disease then existing, to decide definitely whether the appendix or the cæcum was the original site of infection.

Since the appendix was generally found in the midst of the inflammatory mass, it was for the most part taken for granted that this structure was the primary seat of the disease, as the following quotation from Ransom's account shows. This observer in the concluding paragraphs of his report writes : " It may be perhaps assumed that in this case the earliest seat of the disease was in the vermiform appendix, that here a fragment, or a whole grain, of corn or grass lodged, and the actinomyces parasite developed on it." This assumption was pointed out by Waring in 1905, when he published the account of seven cases of the disease in this region, and championed the view of the cæcal origin of the disease. The ascribing of the primary lesion to the appendix was at those times somewhat of the nature of a speculation, since no really minute pathological data were produced in support of such a proposition.

On the other hand, the view that such infection may at times start in the wall of the cæcum is borne out by indisputable pathological evidence.

In Chiari's case, which gave no symptoms during life, a small ulcer 1 cm. in diameter and 5 mm. thick was found post mortem in the cæcal mucosa. It was covered by a greyish layer, which in films and culture showed the presence of *Actinomyces bovis*. Again, in 1920 Slesinger reported a case of an ulcer in the wall of the cæcum which perforated into the general peritoneal cavity. From this case also a culture of the same organism was obtained. Such early stages of this infection within the abdomen are not often met with, but they afford ample proof that the initial lesion is at times in the wall of the cæcum itself.

In the seven cases which Waring reported, the disease was of the chronic nature mostly then described. In the two post-mortem records in this observer's series, an ulcer was found in the cæcum ; but in neither appendix nor ulcer could the ray fungus be demonstrated, although it was present to naked eye and microscope in the retroperitoneal tissues and liver.

In view of such absence of the organism in these ulcers one is forced to raise the question whether the ulcer was the primary lesion or secondary to the inflammatory reaction in the intestinal wall due to the extensive retrocæcal disease. In the post-mortem of Ransom's case it is expressly mentioned that no lesion of the cæcal mucosa was found ; neither was the organism of actinomycosis found in microscopical sections of the walls of cæcum or appendix.

It appears to me that the logical deduction from these consistently negative findings in such cases is that the ulcers in the cæcum which have been described by Waring as

the primary lesions are only of a secondary nature as far as they are concerned in the etiology of the condition.

The other view, which is upheld by Kelly, Cope, and others, maintains that the lesion commences in the cæcal appendix. The possibility of this occurrence is not based on such accurately ascertained pathological facts as is that of the former view. On this subject of actinomycosis of the appendix Battle and Corner write: "As this organ is so situated that it represents the back-water and sample culture tube for the cæcum, which is the first resting-place of the products of digestion after leaving the stomach, it would be surprising if actinomycosis was not found here." This statement may sound more or less true, but accurate pathological knowledge cannot be based on human expectations. More and more records are appearing in the literature, similar to the four cases recorded here, in which a sinus yielding *Actinomyces bovis* has persisted or appeared soon after an operation for acute, subacute, or chronic appendicitis.

In previous times, with the expectant treatment of appendicitis, such cases were left until tumour formation brought the sufferers under the surgeon's hand. It is on such cases that the view of the appendix being the seat of the primary lesion is largely based. Such opinion maintains that an actual lesion in the appendix mucosa was due to the ray fungus, and the infection has persisted after removal of the offending member. With this view of the exact pathological sequence of events at the onset of actinomycosis of the right iliac fossa I cannot quite agree, after a perusal of the recorded cases in the literature, and after investigating the four cases set forth at the end of this paper.

Now, wherever *Actinomyces bovis* attacks the body, whether in the cervicofacial region, thorax, or abdomen, it is of a chronic nature with its own peculiar features. There is a proliferative or neoplastic change, which is followed sooner or later by softening and pus formation in the tissues immediately surrounding the fungus. Clinically, two types have been described as occurring in the ileocæcal region: the indurated or neoplastic, and the soft. Pathologically these are one and the same, it being only the time factor as regards the date of softening which differentiates them in clinical descriptions. Since the fibrous or 'woody' formation is essentially the result of a protective reaction, it is quite to be expected, as is observed, that the soft type offers a worse prognosis than does the indurated.

The most characteristic feature found in the tissues which are the seat of actinomycotic infection is the marked proliferation in the connective-tissue cells before the stage of softening is reached.

When the disease has been discovered in an abscess or sinus following an appendicectomy, the case has been labelled and reported as one of actinomycosis of the appendix.

Waring, in putting forward the case for cæcal origin, has drawn a parallel comparison between this infection of the ileocæcal region and an infection of the same parts with the bacillus of tuberculosis. He points out that in the latter infection the disease in the appendix is usually secondary to a primary lesion of the cæcum. It must be borne in mind, however, in making such a comparison, that the lesions peculiar to the bacillus of tuberculosis have been demonstrated in the walls of the appendix, as has also the actual presence of the specific organism of the disease. Now, in none of the recorded cases of so-called appendicular actinomycosis have the characteristic lesions produced by this infection been described as seen in the appendix itself. I can find no record of microscopical evidence of the presence of the organism of actinomycosis in the walls of the appendix. In the post-mortem records of Ransom's case, as well as in those reported by Waring, this absence is noted. Gaylord and Aschoff, whilst describing the pathological histology of the appendix, definitely call attention to this fact, as is seen in the following quotation from their work: "A few cases of actinomycosis have been described. The wall of the appendix in these cases shows simple inflammatory changes, the organisms being first detected in the perityphlitic accumulation of pus."

If the appendix in these cases is actually the seat of the primary lesion, it is reasonable to expect that some evidence in the tissues of the organ should be present. To a possible argument that the organism has been discharged in the process of softening in the primary

focus, I would point out that the appendix of *Case* 4 in this series was submitted to an exhaustive scrutiny at a very early stage of the disease, and that the results here were also negative.

In cases where a sinus yielding sulphur granules has persisted, the appendix does not differ in appearance from appendices removed from patients who heal and recover without such complications. The four cases which are reported in this paper were all patients who presented the signs and symptoms of typical acute appendicitis of comparatively short history; the longest, of five days' duration, in *Case* 4. At the time of removal there was nothing, save in this last case, to lead one to suspect the presence of such infection. In the first three cases the appendices were opened and examined at the time of operation, but were destroyed without bacteriological or microscopic examination. They presented no signs apart from those usually seen in acutely inflamed appendices. In these cases a sinus had persisted for some time before a really energetic search was made, which resulted in the discovery of the typical sulphur granules. In *Case* 4, as the history shows, the nature of the infection was settled beyond doubt before removal of the appendix. The knowledge gained from previous experience allowed one to place a correct interpretation on the clinical findings observed at the first operation. In consequence, a careful and persistent search resulted in the discovery of the infecting organism at an early date. The appendix was therefore carefully preserved and examined in section, as was the portion of the cæcal wall which was removed with it.

The appendix of this case had the appearance commonly seen in a patient who gives a history of repeated attacks which finally culminate in a reaction sufficiently acute to necessitate immediate operation. It was not markedly thickened. and, on making a longitudinal section of the organ, a stricture, dating from one of the previous attacks, was present about an inch from the tip. The mucous membrane was inflamed, especially in the distal half. There was no sign of ulceration, but the portion beyond the stricture was slightly bulbous and distended with a turbid serous fluid. In this were several sulphur granules which, to the naked eye, in film, and in culture, were undoubtedly those of *Actinomyces bovis*. Although the organ was most carefully sectioned, no sign of this organism or of the characteristic tissue changes which it gives rise to could be seen in the mucous membrane or other layers of the wall of the appendix. The appearance was that of a simple inflammation with a large preponderance of eosinophil cells, which Eastwood showed is found in appendices in which inflammatory reactions have been present for several days.

Here, then, was a case in which the organism of actinomycosis was present in the lumen of the appendix, causing, as far as our pathological findings show, no trouble. Further, neither the infection nor its significant tissue changes could be found in the walls, whilst outside, in the connective tissue of the retroperitoneal area, the organism was present as granules which were giving rise to the typical changes of proliferation and softening. In the post-mortem descriptions of Ransom, Waring, and of *Case* 1 of this series, the main spread of the disease was in the muscles and connective tissue of the posterior abdominal wall. In some cases it has spread down into the hip-joint, whilst in others, as in *Case* 1, it has caused extensive destruction of the bodies of the vertebræ. The lymphatic glands were not affected by the organism of actinomycosis; any enlargement or suppuration in these tissues was the result of the secondary infection which is invariably present in the disease in this situation.

The occurrence of pyæmia in actinomycotic infections is described as rare. In other sites of the infection this is so, though one or two cases have been reported. In the ileo-cæcal region, however, it occurs frequently, when it is a definite portal pyæmia. Most of the post-mortems recorded describe multiple abscesses in the liver filled with the yellow granules of the ray fungus. The microscopic examination of such foci shows that they commence near the portal spaces.

In attempting to solve the question of the etiology of this condition observers have rather tended to sum up without completely weighing all the evidence. At first sight the histories and post-mortem findings of Ransom's, Waring's, and similar cases, together with

the undoubted lesions of the appendices in my four cases and in the many other recorded cases in recent literature, seem strong evidence that we must look to the appendix rather than to the cæcum for enlightenment on the early pathology of the infection of these regions with *Actinomyces bovis*. The view that the primary lesion due to the ray fungus is present in the appendix is not, I maintain, borne out by investigation. The part which is played by the vermiform appendix is a purely secondary one.

J. W. Keefe expresses the opinion that an acute or chronic inflammation of the appendix may open the door to the actinomycosis organism. Such is, I believe, the true etiology of these cases. The primary condition is that of an ordinary attack of appendicitis, in which the damage done to the walls of this organ by such inflammation has allowed the *Actinomyces bovis*, which was previously a harmless inhabitant of the lumen of the intestine, out into the retroperitoneal tissues, where it finds its best nidus in which to play havoc with the health, and oftentimes with the life, of the patient.

It is noteworthy that in all these four cases, as in others so recorded, the appendix, either wholly or for the greater portion of its course, occupied an extraperitoneal position behind the cæcum. In cases where the appendix is in the peritoneal cavity, this infection, if it occurs, first manifests itself as a mass in the wound of the anterior abdominal wall. Here it is clearly a case of wound infection at the time of operation. The fact that this condition in the right iliac fossa does not follow the cases of usual intraperitoneal position of the appendix can be explained by the extraordinary resistance shown to this infection in all cases by the peritoneal membrane. It is only in the last stages, when the patient's resistance is hopelessly broken down or when a secondary abscess in the liver has ruptured, that we see the defences of the peritoneum fall before the infection of *Actinomyces bovis*.

Such an explanation of the etiology of these cases at once raises the question of the actual character, the mode of entry, and the natural habitat of the infective agent which is responsible for actinomycosis in man. Traditions, even though not well borne out by modern knowledge, often die hard. This is true in the medical world just as in other branches of human activity. It is only in recent times that any general scepticism has arisen with regard to the theory that infection was borne by corn, barley, or grass. So much was this view held and promulgated that, if actinomycosis was found in any patient, suggestions were almost universally put to him that his habits or occupation caused him to chew raw corn or to carry straw or like material in his lips whilst at work. There is really no sound evidence for looking on actinomycotic infections as being a prerogative of rural occupations. In my own four cases there is no connection between patients and agricultural habits. In all instances they were town dwellers with work which confined them indoors. In other series of reported cases there is by no means a preponderance of rural inhabitants amongst the sufferers. There are certainly a number of cases described in which a blade of grass or an ear of corn has been found in connection with the lesion. There is, however, no actual evidence to prove that such foreign body carried the infection or that it served any function in the production of the disease other than that of producing the necessary lesion of the mucosa.

The organism which is found in such foci in man, and which was discovered in the four cases of the present series, is the *Actinomyces bovis* first described by Wolff and Israel, and afterwards re-investigated and verified by Wright. There is no record to show that this organism has ever been found outside the animal body. It is an anaerobic streptothrix which will not grow on all media and which fails to reproduce itself and soon dies at room temperature. In animal inoculation experiments it gives rise to lesions containing the typical yellow granules, though it has not yet been possible to reproduce the typical 'woody' formation found in actual disease. The organism described by Bostroem, together with those obtained from grasses and corn, have always failed to give positive inoculation results in animals. In addition these latter organisms are aerobic and grow freely on all media at room temperature. On account of the cultural characteristics of his organism and those of Wolff and Israel, Wright does not admit its existence outside the body, although he has no actual proof, apart from its nature in test-tube and in inoculation experiments, to substantiate this view. Lord has shown that this organism does exist in

the throat and teeth cavities of persons who exhibit no sign of the disease. In his researches typical experimental lesions were obtained in guinea-pigs.

In view of such evidence, those who maintain the source of infection to be present in corn have fallen back on a theory of the possible dual form of existence of the organism, with characteristics which differ when present in the animal body from those observed when outside in the vegetable world.

At present the most we can say is that, though the life-history of this organism is but imperfectly understood, the evidence is accumulating to support Wright's view of alimentary habitation. One argument against this opinion which must certainly be considered and met in future investigations is the comparatively few cases of the disease, and the entire absence of any recorded case of post-operative infection occurring in the wide realm of abdominal surgery.

DIAGNOSIS.

The diagnosis of this infection can, of course, only be made with certainty after discovery of the sulphur granules of the *Actinomyces bovis* in the pus from abscess or sinus. In the cases which follow appendicectomy for an acute or subacute condition, it is well-nigh impossible to diagnose the infection at the time of operation. At this stage one would have to examine systematically the contents of every appendix removed for this specific organism. Even then the difficulties of such detection are so numerous, that one cannot have enough hopes of success to justify the time expended.

The appearance of a mass in the right iliac fossa or in the actual scar of the operation should at once make the surgeon suspect the presence of this infection. It is no uncommon practice, as records show, for such a case to be re-explored with the fear that a swab has been left behind. An incision reveals a hard indurated mass which may or may not at this stage deliver the typical yellow granules.

In the patients who appear before us at a later stage with marked tumour formation there are certain clinical features which at least should make us strongly suspect the true nature of the condition. The patient is more usually of the male sex. The sex ratio in the four cases of this disease here reported is three males to one female, which is the proportion that Osler worked out in this infection. The patient has usually had pain of a varying degree in the right iliac fossa for some few days. He will complain of exacerbations of this pain on moving the right lower limb, especially on mounting a vehicle or on going upstairs. This feature of the pain is so characteristic of this particular infection in the ileocæcal region as to prove a strong foundation stone on which to lay an exact diagnosis. The temperature and pulse are not as a rule much raised, though these vary with the degree and virulence of the secondary infection already present.

On examining the patient the right thigh is held flexed at quite an appreciable angle, whilst any attempt to extend it, whether of a passive or of an active nature, causes excruciating pain. The abdomen moves fairly well, the peritoneal cavity itself being free from infection. The main feature of the tumour is its characteristic hardness, which at times may even suggest a malignant neoplasm. Such diagnosis is as a rule put out of court by the short history of the case, the definite signs of inflammatory reaction, and the rapidity of tumour formation, with obvious involvement of the psoas muscle. One pitfall in diagnosis is to miss a primary new growth of quite small dimensions in the right testis with secondary deposits in the glands in this region. On exploration, the hard woody nature of the tissues around the appendix lying behind the cæcum should make us morally sure of the true nature of the case. The ultimate confirmation of the diagnosis must wait for the finding of the sulphur granules.

It is essential for the welfare of the patient that the true nature of the infection should be proved at the earliest possible opportunity. It is often the case that the granules are not found in the primary abscess or even on several subsequent examinations. Repeated and regular searches are necessary in many cases before success crowns our efforts, since granules are only discharged in the pus when an area of softening gives way and

yields up its contents. It is often far from easy to clinch the diagnosis of these cases : it requires patience and care. On one point I feel most strongly : the proper person to look for the ray fungus is the surgeon of the case or his own assistant. The easiest and most certain method is to allow the discharge from the sinus to run down the side of a sterile test-tube whilst dressing the case. If the sinus does not yield sufficient for this, gently curette the walls of the track with a spoon, and allow the blood which escapes to flow in a similar fashion. The granules will cling to the sides, allowing the liquid portion of the pus to run to the bottom of the tube. They can then be picked out and examined microscopically. If all cases were so dealt with, the diagnosis could be settled far sooner than is often the case. Too frequently the pus is kept and allowed to clot before reaching the bacteriologist. It must be remembered that it is essential for diagnosis and culture that the actual yellow granules be found and isolated. They alone bear the significant club formation. This is not easy if the pus has clotted, and the streptothrix soon dies at room temperature, so that the chances of thus finding it are greatly diminished, as experience often proves.

TREATMENT.

The treatment of actinomycosis in the region of the right iliac fossa is of a more exacting and anxious nature than is the case with the same infection in other situations of the body. The condition is here complicated by the extent and variety of secondary infections which are invariably present, and also by the wide extent of the infected area when the diagnosis has been definitely proved.

The first important line of treatment with large doses of potassium iodide should, I am sure, be started as soon as the clinical features warrant even the suspicion of the true nature of the case. As regards operative measures, it is clear, I think, that the condition is one in which the surgeon who holds his hand gets the best results. At first sight the prevalence of secondary abscesses in the liver of a definite pyæmic origin would, perhaps, be in favour of an extensive intestinal excision as soon as diagnosis is certain. Such an operation is bound to carry in its train a large degree of risk. Also, there is no disease found post mortem in the intestine : the infection is a retroperitoneal one and invades the portal system from behind only at a late stage of the disease. Gangolphe and Duplant, in their review of this condition, are strongly against any form of resection. The rapidly fatal result in *Case* 4 is much against such treatment. Any form of resection or extensive curettage is, I am certain, strongly to be deprecated. In cases where the patient is first seen with a definite tumour formation no attempt should be made at appendicectomy. The appendix is not actually the seat of disease, and protective layers are broken down and fresh ground is thrown open to the infection by any attempt to remove it. The activities of the surgeon should be limited to ensuring free drainage when abscess formation and softening occur. Hydrogen peroxide used as an irrigating fluid is, I think, the best local application. It also helps, more than any other disinfectant, to diminish the peculiarly foul odour which arises from actinomycotic infection in this neighbourhood. The smell when once recognized is peculiar to such conditions; perhaps Waring's description of an odour like sulphuretted hydrogen gives the best idea which words can convey. It is particularly offensive, penetrating, and clinging.

Medical treatment consists chiefly in the administration of increasing doses of potassium iodide until the patient is taking 100 gr. three times a day. When this dose is reached many authorities advise that the drug should be discontinued periodically for a few days to allow softening to take place, and thus ensure a more certain effect of the drug on the infective organism. Some have advocated the use of *x* rays in cases of this infection. This form of treatment was used in *Case* 3 of this series, but with disappointing results. Heyerdath has reported successful results from the use of radium. But here, and in the reported improvements under the application of *x* rays, the lesions have all been in the cervicofacial region where one's efforts are not crippled from the start, as here, by a foul secondary infection. Some observers are high in their praises of the results of

autogenous vaccines. Here again, the same must be said of these successes as was mentioned with regard to *x* rays and radium. All four patients here reported were treated with autogenous vaccines, both for their actinomycosis and secondary infections. The disappointing results of such treatment are shown only too clearly by the end-results of these cases.

PROGNOSIS.

The present prognosis of actinomycosis of the right iliac fossa can only be described as gloomy in the extreme. The average mortality of recorded cases of abdominal infection of this nature is somewhere in the neighbourhood of 80 per cent. In Waring's series 3 cases only died out of a total of 7. This mortality of 42 per cent is much lower than that of any other observer. In the cases reported by Colebrook the mortality-rate was 83·3 per cent. The results published by Mattson are the same as those of the present series, where the death-rate from the disease is 100 per cent.

The length of time which intervenes between the first symptoms and death varies from six months to two years. There is no doubt that the life of the patient is of longer duration in those cases where conservative surgery has played a part. It was in a large measure the main factor responsible for the lower mortality in Waring's series. On the other hand, the rapid end of *Case* 4 in this series speaks ill for the results of any extensive surgical endeavours.

Until the life-history of the infecting agent has been more fully worked out, so that measures of prevention may be adopted, the only hope of reducing the present high mortality of this disease must lie in earlier diagnosis and the subsequent possibility of prompt treatment.

REPORTS OF FOUR CASES OF ILEOCÆCAL ACTINOMYCOSIS.

Case 1.—Miss F. T. Age 20. Town-dweller. Shop assistant.

In the early part of May, 1920, the patient complained of pain in the right side of her abdomen. Treated by her doctor with fomentations. Pain rapidly disappeared, and patient was soon about again. Some sixteen days later sudden acute attack of abdominal pain. Vomited several times. Temperature 100°; pulse 110. Tenderness and rigidity in right iliac fossa.

May 20, 1920.—Operation: appendicectomy through a pararectal incision. Appendix gangrenous and perforated lying behind the cæcum surrounded with pus. Drained through abdominal wound and a slab incision in loin. *Aug.* 3.—Sent to convalescent home with sinus in loin. *Oct.* 16.—Large abscess in loin opened and drained. *Nov.* 7.—Discharged healed. *Dec.* 14.—Another abscess drained through the loin. A discharging sinus persisted. *Feb.* 10, 1921.—Actinomyces found in discharge from sinus. Growth obtained after method of incubation described by M. H. Gordon. Vaccine prepared. Doses from 25 million to 500 million given at weekly intervals. Potassium iodide administered internally. *July* 17, 1921.—Death.

Post-mortem Findings.—Body extremely emaciated. Considerable post-mortem discoloration of abdominal wall. Several gangrenous sores on back. Old operative scar over site of appendix. Apertures of four old sinuses into right loin, showing marked pouting of granulations.

Right lung.—Collapsed, especially in upper lobes. Pleura adherent at right base where pus had spread through the diaphragm from abdominal condition. No secondary abscesses. *Left lung.*—Normal. *Heart and Pericardium.*—Nothing abnormal discovered beyond some fatty changes in heart muscle. *Vertebræ.*—Eroded in upper lumbar and mid-dorsal region by extension of disease. *Spleen.*—Showed amyloid changes. *Peritoneum.*—Large quantity of pus present behind the peritoneum on the right side, tracking up behind the right kidney and beneath right cupola of the diaphragm. Peritoneum much thickened and in parts necrotic. *Intestines.*—Very adherent on right side. Cæcum showed no macroscopic changes. Appendix absent. Healthy scar in wall of cæcum where stump was invaginated. *Right kidney.*—Surrounded with pus. Capsule stripped easily. Amyloid changes present, as was the case in the left renal organ. *Liver.*—Marked fatty changes present. Advanced degree of perihepatitis. Adherent to diaphragm on right side. No secondary abscesses.

Case 2.—C. D. Age 21. Male. Town-dweller. Clerk.

Pain in the right iliac fossa for two days before admission.

Aug. 31, 1920.—Appendicectomy through gridiron incision. Appendix perforated behind the cæcum. Small amount of pus present. Abdomen closed without drainage. *Sept.* 27.—Discharged. *Oct.* 8.—Tender swelling to outer side of operation scar. *Oct.* 21.—Swelling explored. No pus found, but a hard mass was incised and drained. It lay to the outer side of and behind the cæcum.

Jan. 25, 1921.—Sinus still discharging. *Aug.* 8.—Seen as out-patient with sinus still present. Actinomyces found in discharge. Vaccine prepared as in *Case* 1. *Aug.* 11.—Sinus opened up. Pus containing granules evacuated. Cæcal wall not involved. Psoas muscle widely infiltrated. Treated with vaccines and potassium iodide. Application of *x* rays Aug. 29, 2x; Sept. 2, 4x; Sept. 5, 4x; through 3 mm. Al. *Oct.* 15.—Discharged with persistent sinus. Potassium iodide continued. Developed signs of amyloid disease, and symptoms of involvement of right lung appeared later. *June*, 1922.—Death. No post-mortem examination.

Case 3.—J. H. Age 37. Town-dweller. Machinist.

Three days' history of pain in right iliac fossa. Vomiting one day.

Oct. 18, 1920.—Acutely inflamed retrocæcal appendix removed. Wound drained. *Nov.* 8.—Discharged with a sinus. *Dec.* 15.—Sinus ceased discharging. *Jan.* 7, 1921.—Swelling in right iliac fossa. Incised. A considerable quantity of pus evacuated. Abscess cavity drained. *March* 7.—Actinomycosis granules found in pus from sinus. Treated with vaccines and potassium iodide. *May* 3.—Discharged with persistent sinus. *Dec.*, 1921.—Death. No post-mortem examination.

Case 4.—T. K. Age 42. Town-dweller. Mechanic.

Pain in the ileocæcal region for five days. Vomiting two days. Right lower limb held in position of flexion for one day.

March 7, 1922.—Operation: gridiron incision. Appendix felt in a hard mass behind the cæcum. Abscess cavity opened and drained. Appendix not removed. Actinomycosis was thought to be the probable infection. Examination of pus obtained at operation proved negative. Pus from wound examined daily. *March* 19.—Discovery of yellow granules. *March* 20.—Wound enlarged. Appendix removed with one inch of cæcal wall at its base. All infiltrated tissue in the region of the psoas muscle removed. Wound drained. Treated with vaccines and potassium iodide. *June* 3.—Death. Post-mortem examination refused.

ADDENDUM.

Since writing the above, I am able, through the kindness of Mr. Graham Simpson, to add the notes of a further case of this condition. This history well illustrates some of the more important features of these cases.

May 10, 1921.—Feverish cold and cough, with pain in the right iliac fossa. *May* 23.—Recurrence. *May* 24.—Temperature 99·8°; pulse 102. Rigor. Tenderness and resistance over appendix. Pain relieved by passage of flatus. Swelling remained, but general condition improved: occasional colicky pains near umbilicus, relieved by passage of motion. *June* 3.—Seen in consultation. Looked well. Temperature 97·6°; pulse 120. Tongue clean: had been vomiting all the night before. *Chest.*—Nothing abnormal; no cough. *Abdomen.*—Large, hard tumour in right iliac fossa; not very tender; quite fixed. Great difficulty in extending right thigh. No dysuria. Appendicitis diagnosed. Recommended waiting till lump had disappeared. *June* 17.—Operation: appendicectomy; very large adherent appendix; no ileal kink. Healed by primary union. *July* 8.—Still some thickening felt in right iliac fossa; otherwise well. *Nov.* 9.—Localized tender thickening in abdominal wall under lower quarter of scar; skin over this red (? foreign body). *Nov.* 10.—Second operation: incision and evacuation of two ounces of pus from granulation-tissue-lined cavity on surface of aponeurosis; no foreign body, no cause found though carefully looked for; drained. *Nov.* 14.—Seen again; quite well; very little pus, but no diminution of thickening. Some time later actinomycosis granules were found in the pus. *Jan.* 1923.—Seen again. Improved under potassium iodide. Sinus still persists.

CONCLUSIONS.

1. Actinomycosis of the right iliac fossa gives a clearer and more accurate description of the condition than does the older nomenclature of ileocæcal actinomycosis.

2. The disease in this situation is more common than is generally supposed. The apparent increase in its incidence can be explained by improved diagnosis.

3. In the past, the large extent of the disease when first seen has masked the original site of infection. In consequence, assumption rather than accurate investigation has sought to settle the point of difference of opinion between cæcum and appendix.

4. Cases of infection of the cæcum have been reported, but no recorded case of a similar condition in the walls of the appendix can be found.

5. The appendix plays purely a secondary part in the etiology of this disease of the right iliac fossa. It acts as a *locus minoris resistentiæ* through which the organism escapes into the retroperitoneal tissues.

6. Conservative surgery gives the least unpromising results. It should be limited to simple incision, evacuation, and drainage. Resection and extensive curettage should play no part in treatment.

7. Large doses of potassium iodide, together with an early administration of autogenous vaccines, offer the best hope of cure.

8. Earlier diagnosis must be made if the results of treatment are to improve and if the present high mortality-rate of 80 per cent is to be reduced.

9. In all the four cases here reported the organism was similar in its cultural characteristics to that described by Wolff and Israel.

I wish to tender my best thanks to Mr. A. Cuff and Mr. Ernest Finch for kindly allowing me to use their cases, and also to Mr. S. Graham Simpson for valuable advice and help in compiling this paper.

REFERENCES.

BATTLE and CORNER, *Diseases of Appendix Vermiformis*, 1904, 164.
COLEBROOK, *Lancet*, 1921, i, 893.
COPE, Z., *Brit. Jour. Surg.*, 1915, iii, 53.
DEAN, C. W., *Brit. Med. Jour.*, 1917, i, 82.
GANGOLPHE and DUPLANT, *Rev. de Chir.*, 1897, 503.
GAYLORD and ASCHOFF, *Pathological Histology*, 171.
GORDON, M. H., *Brit. Med. Jour.*, 1920, i, 435.
GRIFFITH, F. W., *Surg. Gynecol. and Obst.*, 1921, xxxiii, 548.
HINGLAIS, *Internat. Clinics*, 1921, iii, 59.
HEYERDATH, *Jour. Amer. Med. Assoc.*, 1919, lxxiii, 1928.
KEEFE, J. W., *Amer. Jour. Obst.*, 1919, lxxix.
LORD, *Jour. Amer. Med. Assoc.* 1910, iv.
MATTSON, W. W., *Surg. Gynecol. and Obst.*, 1922, xxxiv, 482.
PONCET and BERAND, *L'Actinomycose Humaine*, Masson et Cie, 1898.
RANSOM, *Med. Chir. Trans.*, 1892.
SLESINGER, *Lancet*, 1920, i, June.
WARING, H. J., *St. Bart's. Hosp. Reports*, 1905, 197.
WOLFF and ISRAEL, *Virchow's Arch.*, 1891, cxxvi, ii.
WRIGHT, *Jour. Med. Research*, 1904–5, xiii.

GIANT-CELLED TUMOURS OF TENDON ASSOCIATED WITH XANTHELASMA.

By ROBERT OLLERENSHAW, Manchester.

In February, 1920, I saw a young Jewess, 18 years of age, who complained of a painful swelling in the region of each heel. I found, in each foot, a hard, well-defined tumour situated over the tendo Achillis about two inches above its insertion. The two tumours were almost symmetrical and are well illustrated in *Fig.* 368. The skin over them was a little darker than the normal skin of the part, and it was thickened from pressure.

Fig. 368.—Bilateral giant-celled tumours of tendo Achillis.

The patient had noticed the slow enlargement of the area for a period of three years, but had not sought any advice until she began to suffer pain from boot pressure. In all other respects the feet appeared to be normal. In the skin of the right arm, just above the inner condyle of the humerus, was a patch of what is known as xanthelasma.

At operation the following note was made: "The tendon was very greatly thickened and had many yellow-coloured areas on its surface and also infiltrating its fibres. Certain of these patches were present also in the subcutaneous tissue. The tendon was trimmed down to a little more than its accustomed size and the skin repaired."

Since that date, nearly three years ago, I have seen the girl on several occasions, and, after she left the district, I have had a recent report from her medical attendant in the South of England. There has been no recurrence of the growth. A second patch of xanthelasma has appeared on the left arm in the corresponding position to the one which existed previously on the right side. The tumours are very interesting and, on section, show among the tendon fibres areas where giant cells are present in considerable numbers. *Figs.* 369 and 370 show low-power microphotographs of sections of the tumours, and *Fig.* 371 shows a drawing made from a portion of the section seen in *Fig.* 369 on a much larger scale. A number of tumours of similar histological characters, but arising from tendon sheaths, have been reported, and have been called sarcoma, myeloid tumour, myeloxanthoma, and various other more or less descriptive names. Targett[1] expressed the view that giant-celled tumours of this type were clinically and histologically malignant in character. Bellamy[2] is of opinion that, although the presence of a large number of giant cells gives the tumour a claim to be called 'myeloid', yet the growth had no real right to be so classed. He regards it as due to the proliferation of the endothelial cells of the blood-vessels, and suggests the name of myeloid endothelioma. Fleissig[3] describes several giant-celled tumours associated with tendon sheaths, and distinguishes them from new growths. He found them in the hand, foot, forearm, and leg below the knee, and stated as his opinion that they were not giant-celled sarcomata but inflammatory granulation growths, and should be called granulomata of tendon sheaths. Tourneux,[4] in 1913, reviewed the literature of 93 cases of sarcomata of tendon sheaths, and found that 54 were of a xanthic type containing giant cells; 8 of these were in the lower extremity, 5 being on the toes and 3 in the malleolar regions. Broders[5] recently investigated 17 cases of this nature which have been seen in the Mayo Clinic at Rochester, U.S.A. He found no recurrence after local removal. In the case which I am here recording the occurrence of the tumour as a bilateral condition points to some constitutional causation rather than to local new growth. The tumour was not one of the type which originates in tendon sheath, as in the cases reported by the foregoing authors. The tendo Achillis has no true sheath, and the tumour was definitely in and of the tendon itself and must have originated there. The presence of the plaques of xanthelasma in other parts of the body supports the probability of a xanthic constituent in the tumour, and the appearance of the many yellow areas infiltrating the tendon, as seen at operation, lends further support to this view. Unfortunately the block of tissue from which sections were cut was lost before any sections had been stained to show the presence

FIG. 369.—Low-power section of tumour.

FIG. 370.—Low-power section of tumour.

of fats. There seems to be little doubt, however, that the material which occupied the spaces, shown so well in *Fig.* 371, round which the giant cells are chiefly grouped, was of a lipoid character, and that the giant cells are to be regarded as being engaged in the removal of this abnormal material.

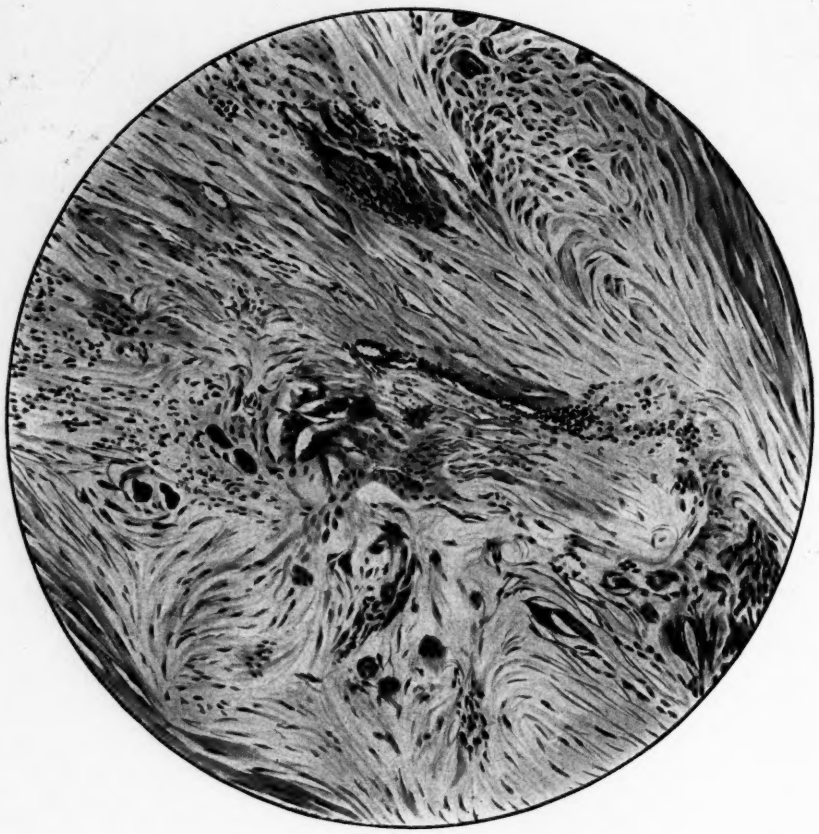


FIG. 371.—Higher-power view of part of section shown in *Fig.* 369.

From the clinical standpoint the question as to malignancy of tumours of this class is of great importance. It appears, from the material before us, that we are thoroughly justified in regarding them as benign, having more the character of a granulomatous change than of a new growth, and that local removal is all that should be undertaken.

REFERENCES.

[1] TARGETT, J. H., "Giant-celled Tumours of the Integuments", *Trans. Pathol. Soc. Lond.*, 1897.
[2] BELLAMY, H. F., "Myeloid Tumours of Tendon Sheaths", *Jour. Pathol. and Bacteriol.*, 1901.
[3] FLEISSIG, J., "Uber die bisher als Riesenzellensarkome bezeichneten Granulationsgeschwulste der Sehnenschieden", *Deut. Zeits. f. Chir.*, 1913.
[4] TOURNEUX, J. P., "Les Sarcomes des Gaines tendineuses", *Rev. de Chir.*, 1913.
[5] BRODERS, A. G., *Collected Papers of Mayo Clinic*, 1919.
[6] STEWART, M. J., and FLINT, E. R., "Observations on the Myeloid Tumours of Tendon Sheaths", *Brit. Jour. Surg.*, 1915.

TUMOURS OF TENDON AND TENDON SHEATHS.*

By ST. J. D. BUXTON, London.

The subject of tumours of tendons and tendon sheaths is of interest owing to the rarity of the tumours. Observations have been made on them since 1860, and in studying the work of these observers one cannot help noticing that the greater part of it has been done by French and German surgeons and pathologists.

TUMOURS OF TENDONS.

Only a few words can be said of tumours of tendons. Primary tumours are very rare and possibly do not occur at all. I can find no trace of a case reported, and in 1907 Ombrédanne[1] agreed with this statement. He was not satisfied that the fibromata and sarcomata described originated in the tendon, but he acknowledged the possibility of osteomata occurring. This condition he compares to myositis ossificans. Tendons, of course, are not uncommonly invaded by direct spread of malignant tumours which have commenced in the bone or connective tissue in their neighbourhood.

TUMOURS OF TENDON SHEATHS.

Our attention is thus practically confined to the tendon sheath. This structure is a specialized part of connective tissue, and is therefore subject to the same neoplasms as are other connective tissues. Such neoplasms are not uncommon ; therefore one searches for some reason why tendon sheaths should be so immune from tumour formation, and it is very difficult to find any facts bearing on this point.

First, our knowledge at the present time of the origin of tumour formation is extremely small.

Next, it has been found that a history of trauma is not uncommon prior to tumour formation. One has no reason to suppose that tendon sheaths are anatomically shielded from trauma; on the contrary, one would suppose that they and tendons were very liable to injury.

In the third place, in all probability chronic inflammation and irritation are predisposing factors to tumour formation. Chronic tenosynovitis is very frequent, and becoming more so with the development of athletics and machinery.

Therefore one is at a loss to account for the rarity of these tumours, especially as, although they are of small size, they cause some limitation of function of hand or foot—a sufficient reason as a rule to make pauper, peer, or physician seek the advice of a surgeon.

The classification of the primary tumours occurring on tendon sheaths is as follows :—

1 *Benign.*—(i) Lipoma ; (ii) Fibroma ; (iii) Chondroma ; (iv) Ganglion.

2. *Benign, but liable to Recur Locally,* and therefore often called locally malignant.—Giant-celled myeloma.

3. *Malignant.*—Sarcoma.

1. **Benign Tumours.**—

i. *Lipomata* are uncommon, and detailed observations have been made on only eighteen cases.[2] Two varieties are recognized. *Lipoma arborescens*[3] occurs, analogous in origin to those from the villi of the joints, which were first described by Müller nearly a century ago. *Lipoma simplex* also occurs. Clinically these two varieties may be

* A paper read before the British Orthopædic Association, October, 1922.

considered together, because the symptoms, signs, locations, treatment, and prognosis are the same.

They are composed of fat tissue without any evident capsule other than the sheath itself to which they are so intimately attached; hence the sheath must be sacrificed in removing the growth. The tumour is liable to surround the tendon within the tendon sheath, and follow it towards its insertion. In these cases there may or may not be fluid within the sheath, and the tendon is intact as a rule. On the other hand, the lipoma may be outside the sheath and attached to it by a pedicle.

They are more frequent on the tendon sheaths of the palm of the hand, but occur also on tendon sheaths in the leg. The commonest symptom is disability, movements with which the tendon is concerned being limited, owing to pain. As with most tendon-sheath tumours, diagnosis has seldom been made before operation, the signs being similar to those of tenosynovitis, simple or tuberculous, which is so much more frequent. The diagnosis of ganglion has been made, and lipoma found at operation. The only treatment is removal, and for their ablation it is necessary to remove a portion of the tendon sheath.

ii. *Fibromata* are about as uncommon as the lipomata, and do not attract any special attention. A specimen of one from a tendon sheath near the ankle exists in the Museum of the Royal College of Surgeons (608). It is the size of a small walnut and lobulated on the surface, and had been growing for twenty years.

iii. *Chondromata* are probably less common than the other simple tumours. Meeting with a case of this kind earlier in the year, my attention was drawn to their rarity. Cartilaginous tumours are rare except in connection with bone. They are reported[4] as occasionally growing in muscles and fasciæ. Erichsen refers to three cases, the tumours being in the tibialis anticus, vastus externus, and pectoral muscles, and Liston removed a chondroma the size of an orange from the vastus externus.

As tumours of tendon sheaths they are considered worthy of mention by Ombrédanne[1] and Delbet.[5] The latter states that they are quite different from true chondromata of bone. Chauvain and Roux[6] report a case in detail, the tumour occurring on the tendon sheath of the extensor tendon of the fourth toe. They diagnosed the case as ganglion, but found a solid nodule attached to the tendon sheath, and had to resect a portion of this sheath to remove it.

Histologically, it was found to consist of hyaline cartilage and fibrocartilage, with bands of fibrous tissue. They consider it to be of the nature of 'traumatic perimusculo-tendinitis', the history being a sequence of a violent blow, followed by ecchymosis, a hæmatoma, and then this tumour.

The history of the tumour in the hand which I report fully is different. The patient was a professional violinist, and she complained of pain and swelling in her left hand for two months, so that she could not play her instrument. She could recollect no definite injury, but this hand she used greatly in playing her violin. There was no doubt about the swelling in the palm. It was in the space between the heads of the third and fourth metacarpal bones, and was soft, non-fluctuant, and did not appear to move with the flexor tendons.

Full flexion of the ring finger was painful, but no limitation of flexion or extension at any of the finger-joints was present. The skiagram showed an area the size of the sesamoid bone at the base of her index finger, situated on the radial side of the neck of the fourth metacarpal bone, which was more opaque than the soft tissues, but less dense than the shadow cast by the bones of the hand. At operation, an encapsuled mass was found on the radial side of the flexor tendons which go to the ring finger. It had a pedicle attached to the flexor tendon sheath, just distal to the metacarpophalangeal joint of the ring finger. The specimen measured $1 \times \frac{1}{2} \times \frac{1}{2}$ inch. It was lobulated, and somewhat grey in colour. The microscopic examination revealed that the tumour was a chondroma. A connective-tissue stroma containing blood-vessels and no abnormal cells surrounded masses of hyaline cartilage. Professor Shattock was kind enough to examine the section, and states that it is a simple chondroma (*Fig.* 372). The opaque area shown in the skiagram is due to calcification of a portion of the neoplasm.

Calcification occurred also in the following case, which is one of Marcus Beck's. Mr. Raymond Johnson[4] refers to it, and tells me he helped Beck remove the tumour from the flexor tendon sheath of the index finger. It was thought to be a ganglion before operation, and the specimen is now in the Museum of University College Hospital Medical School. Mr. E. K. Martin has re-examined the specimen, and owing to his kindness I am able to illustrate a section of the tumour, which was the size of a cherry. It is, like the last, composed of several lobules of cartilage, and there is an area of calcification (*Fig.* 373).

FIG. 372.—Chondroma of tendon sheath. (× 85.)

A specimen (1569, Old Catalogue) that is in the Museum of King's College Hospital shows several tumours on a tendon sheath. The catalogue states that these are fibromata. Re-examination shows them to be composed of fibrocartilage, and it is to be noted that these tumours are multiple, and less lobulated than the previous two which were examined (*Fig.* 374).

In regard to the origin of these tumours, one would suppose that cells capable of producing cartilage had strayed from the primitive basis of the bone into the attached connective tissue. Nothing in the microscopic sections suggests that they are other than benign tumours. There is no evidence to show that they recur after removal.

FIG. 373.—Chondroma of tendon sheath, showing calcification. (× 85.) This section was prepared and re-examined by Mr. E. K. Martin.

(iv.) *Ganglion.*—This condition should be regarded as a tissue degeneration rather than a cystic tumour. In addition to the favourite site on the dorsal carpal region, ganglion may appear in the palm near the metacarpophalangeal articulations, the site of the greater number of tumours of tendon sheaths. It is interesting to note that a ganglion may form inside a tendon.[7] This is certainly a point in favour of the pathology of the formation of ganglion worked out by Ledderhose,[8] and confirmed by Thorn[9] and Franz,[10] being correct. They state that their origin is in colloid degeneration of hyperplastic peritendinous tissues. Histologically a ganglion is indistinguishable from a lymphangioma, but it is not easy to conceive the idea that this condition is derived from dilated lymphatics.

2. **Giant-celled Myeloma.**—This is without doubt the commonest tumour of the

tendon sheath, and has attracted considerable attention, as its pathology is of great interest.

The common site is over the metacarpophalangeal articulation in the palm; but the forearm tendons are not exempt, any more than are the tendons passing over the ankle-joint. There is usually a history of the part having been injured. The tumours are small, growth is slow, and interference with function is late. They are adherent neither to bone nor to skin. The differential diagnosis is very difficult owing to the lack of special signs or symptoms produced by these and other tumours, by which they could be distinguished from tenosynovitis. Tourneux[11] discusses the diagnosis at considerable length in excellent papers, but acknowledges that even should a neoplasm of the sheath be diagnosed, the only evidence to suggest myeloma is small volume, soft consistence, and slow growth. The majority of cases described have not been diagnosed until a section of the tumour has been cut. The growth is lobulated, and has a slight brownish or pinkish coloration—not the grape-juice colour of the myeloma of bone. Microscopically, it is found to be divided into lobules by strands of fibrous tissue. In the lobules are a variety of cells, including multinucleate giant cells, and pigment.

FIG. 374.—Fibrochondroma of tendon sheath. (× 85.)

Broders[12] describes the histology in detail in a recent paper, illustrated with microphotographs. There is evidence that these tumours recur locally, but they must be distinguished from the sarcomata, which, in addition to local recurrence, may form metastases. The distinction is only possible in an early case with the aid of the microscope.

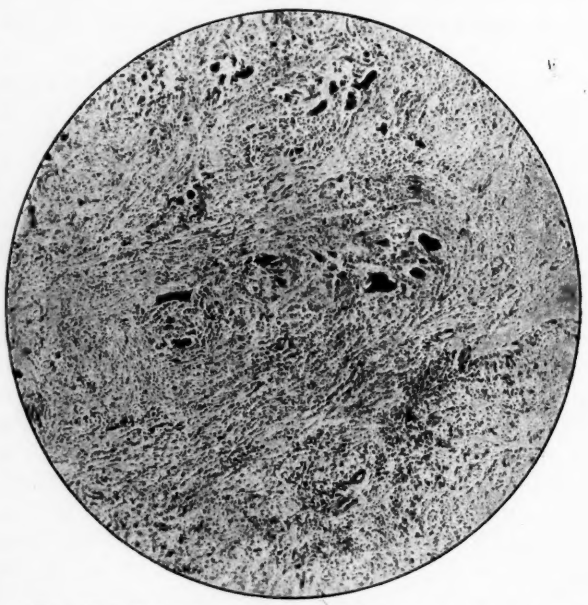


FIG. 375.—Giant-celled myeloma of tendon sheath. (× 85.)

The case I have to report is that of a girl who was hit on the back of the right thumb when playing hockey in November, 1921. There was what she calls a 'gash' over the back of the thumb. In December she noticed a small swelling in the same place which gave her no pain, but a dull ache. It has grown slowly. In September, 1922, ten months later, she came for treatment. It was found she had a small swelling, a little larger than a pea, over the radial side of the first phalanx of the right thumb. It was free of the skin—which seemed to be normal—and was movable laterally. It did not appear to be fixed to extensor or flexor tendon when either was brought into action. There was a small nævus over the side of the terminal phalanx of this thumb.

The tumour was excised under local analgesia. An encapsuled lobulated tumour was removed from the connective tissue deep to the skin. The capsule was attached to the sheath of the long extensor of the thumb and to the tendinous expansion. It felt of solid consistence after removal.

The section shows the tumour to be a giant-celled myeloma, with the typical giant cells in large numbers, as is the case with these tumours (*Fig.* 375).

At the Royal College of Surgeons there is one specimen only of a giant-celled myeloma of a tendon sheath (1608–1). It was excised from the flexor aspect of the right thumb, opposite the interphalangeal joint, of a woman, age 24, a clerk. She noticed the swelling when it was the size of a split pea, two years previously.

A portion of the tendon sheath had to be removed in order to free the tumour, which was 1 inch in length and somewhat oval in shape. Its surface is broken up into many lobulations, and is yellowish in the prepared specimen. On section, the deeper portion is white.

The history of the myeloma of tendon sheaths is one of pathological controversy.[13] The first problem was settled by Heurteux,[14] who separated the myeloma from the sarcoma by the clinical results of the cases. The second problem is as to the exact nature of the myeloma, and this is not yet decided to the satisfaction of all. Professor Shattock tells me that he regards the giant-celled myeloma of tendon sheaths as of the same kind as the giant-celled myeloma occurring at the ends of bones. It is suggested that it is due to the embryonic displacement of bone elements into the tendon sheath, which is continuous with the bone. The elements displaced are those related to the marrow. Tourneux supports this theory. The tumours occur in babies as well as in people of 80, and possibly the appearance of the tumours in babies lends support to the theory. This being so, the chondroma and myeloma of tendon sheath are comparable in their origin, but neither tumour has a connection with the bone.

Stewart, who has done a considerable amount of work on these tumours, considers that they should be called myeloid endotheliomata, as suggested by Bellamy.[15] He believes the myeloid giant cells to be of tumour origin and an essential part of the growth, but that the minute structure is that of an endothelioma, and that cells of inflammation are not present in the tumour. He points out that too much stress has been laid on the presence of pigment and xanthoma cells, particularly by Dor[16] and Fleissig,[17] whereas these cells occur in other tumours and are dependent on tissue disintegration.[18]

The American school, represented by Ely[19] and Broders, find that local extravasation of blood is an almost constant predecessor of the tumour, and regard the growth as a granuloma rather than a neoplasm. The giant cells are accounted for by being regarded as a collection of nuclei surrounded by an irregular mass of hyaline and faintly granular material of foreign-body origin.

3. **Sarcoma.**—This tumour of tendon sheath differs from the giant-celled myeloma in its clinical characteristics, just as the myeloma and sarcoma of bone differ. It is, in fact, a malignant tumour. It starts with local symptoms, the tumour generally being in the line of the tendon, and hard and lobulated on the surface. Sometimes the movements of the joint near the tumour are limited. Pain is not great and is variable. It occurs most commonly, I gather, in the palm of the hand, in the forearm, in the sheath of the peronei muscles, or in the tendo Achillis. Skin attachment is unusual; infiltration into surrounding tissue is the rule. The rate of growth is rapid when the round cell is predominant. Dissemination occurs as in other sarcomata, and the lymphatics are not commonly invaded. If the tumour is removed locally, recurrence follows. Hence amputation of the limb is advised, unless radium or Coley's fluid appeals to the surgeon, as the prognosis is unhappy in these cases. A specimen at the Royal College of Surgeons (No. 607*A*) is that of a chondrosarcoma by the side of the tendo Achillis. This occurred in a girl of 17, and the limb was removed by amputation. The histology shows the growth to be composed mainly of cartilage in which are scattered many irregular foci of calcification. Some of these foci become directly transformed into bone. The matrix of the cartilage is in places fibrous, and bone is likewise developed from such by the

intramembranous method, and the formation of osteoblasts. Here and there groups of branching channels, varying in diameter, have arisen from mucinoid degeneration of the strands of connective tissue, which ramify in the cartilage.

In certain areas the cartilage is replaced by connective-tissue cells in close collections, and the tendon itself is infiltrated by similar cells. There is thus no doubt about its nature, but whether it started in the sheath or tendon we cannot say.

CONCLUSIONS.

1. Primary tumours of tendons are extremely rare, and probably do not occur.
2. The giant-celled myeloma is the commonest tumour of the tendon sheath.
3. The differential diagnosis between tenosynovitis and tendon-sheath tumours presents great difficulties.
4. The giant-celled myeloma—(*a*) Grows as a rule at the site of an injury; (*b*) Has the characteristics of a benign tumour, but is liable to recur locally; (*c*) Is comparable pathologically to the giant-celled myeloma of bone.

The kindness and help afforded me by Professor Shattock and others has been invaluable, and I wish to express my gratitude to them.

REFERENCES.

[1] Ombrédanne, *Nouveau Traité de Chirurgie*, ix, 1907, Baillière.
[2] Strauss, A. *Surg. Gynecol. and Obst.*, 1922, Aug.
[3] Billroth, *Allg. Chir. Path. u. Therap.*, 3rd ed., iii, 641.
[4] Choyce, *System of Surgery*, i, 389.
[5] Delbet, *Progrès méd.*, 1920, xxxv, 462.
[6] Chauvain and Roux, *Bull. et Mém. Soc. Anat. de Paris*, 1920, xc, 75.
[7] Borchardt, *Arch. f. klin. Chir.*, lxii, 43.
[8] Ledderhose, *Deut. Zeits. f. Chir.*, xxxvii.
[9] Thorn, *Arch. f. klin. Chir.*, lii, 593.
[10] Franz, *Ibid.*, lxx, 953.
[11] Tourneux, *Rev. de Chir.*, 1913, xlvii, 817; *Progrès méd.*, 1920, xxxv, 315.
[12] Broders, A. C., *Collected papers of the Mayo Clinic*, 1919, xi, 1032.
[13] Stewart, M. J., and Flint, E. R., *Brit. Jour. Surg.*, 1915, iii, 90.
[14] Heurteux, *Arch. gén. de Méd.*, 1891, i, 40.
[15] Bellamy, *Jour. Path. and Bact.*, 1901, vii, 465.
[16] Dor, *Cong. Franç. de Chir.*, Paris, 1898, session xii, 553.
[17] Fleissig, *Zeits. f. Chir.*, 1913, cxxii, 237.
[18] Stewart, *Jour. Pathol. and Bacteriol.*, 1915, xix, 305.
[19] Ely, *Ann. of Surg.*, 1918, lxviii, 426.

MYOSITIS OSSIFICANS AND VOLKMANN'S PARALYSIS.

NOTES ON TWO CASES ILLUSTRATING THE RARER COMPLICATIONS OF SUPRACONDYLAR FRACTURE OF THE HUMERUS.*

By W. ROWLEY BRISTOW, London.

A consideration of the two cases here reported is of interest in illustrating two of the rarer complications of fractures about the elbow.

1. Traumatic Myositis Ossificans.

The child, a girl of 6½ years, sustained a supracondylar fracture by a fall in June, 1921. The local doctor reported that he had reduced the deformity under anæsthesia, and that the next day massage and mobilization had been commenced. (*Fig.* 376.)

For a time, all is reported to have gone well; but two weeks after the accident, the elbow became less movable, and the child complained of severe pain on any attempt at movement.

Fig. 376.—Skiagram taken June, 1921.

I first saw her three weeks after the accident, and, on examination, the region of the elbow was hot, inflamed, and tender. The whole area was hard and indurated, and only a few degrees of movement were permitted, the elbow being practically fixed at a right angle. A lump could be made out in front of the elbow, and *x*-ray examination (*Fig.* 377) revealed new bone formation, presumably in the brachialis anticus. The arm was put at rest in a 'collar and cuff' support, and all physical treatment discontinued. The 'collar and cuff' consists of two loops—one surrounding the neck and the other the wrist—joined together by a length of bandage, so holding the elbow at any required angle. This contrivance was originally described by Hugh Owen Thomas under the term 'gauge halter'.[1]

After leaving the elbow at rest in this support for a few days, it was possible to bring it gradually into the flexed position by shortening the connecting bandage: no reaction followed the manœuvre, which took some five or six days to complete.

A skiagram taken on Aug. 30 (*Fig.* 378) shows the shadow of the new bone to be smaller, but more opaque and defined. All inflammatory signs had subsided, and the elbow was allowed to drop by degrees to a right angle, by adjustment of the sling, without causing pain or local reaction, the power of voluntary flexion being well retained.

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* A Paper read at the British Orthopædic Association at the Royal Society of Medicine, on Friday, October 20, 1922.

On Nov. 16, five months after her accident, movement was free, controlled, and painless, from full flexion to a right angle—the degree allowed by the sling. The *x*-ray

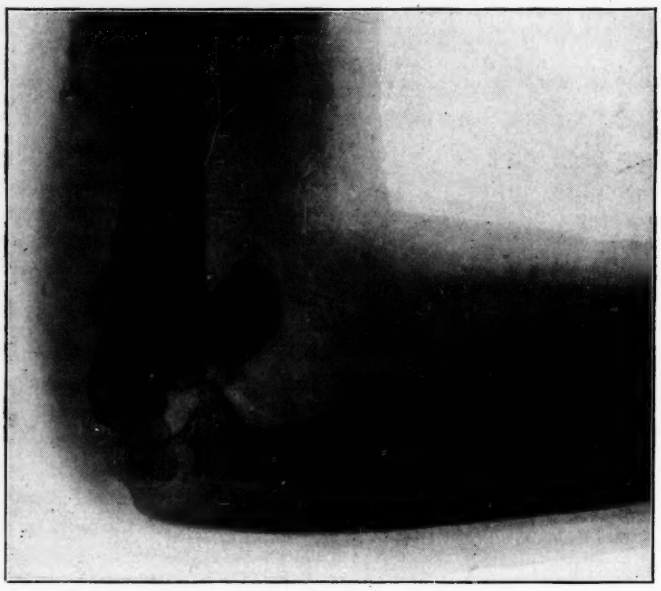

FIG 377.—Skiagram taken on July 17.

showed a great decrease in the new bone (*Fig.* 379). Free movement of the joint was allowed from this date, but no passive or forced movements were permitted.

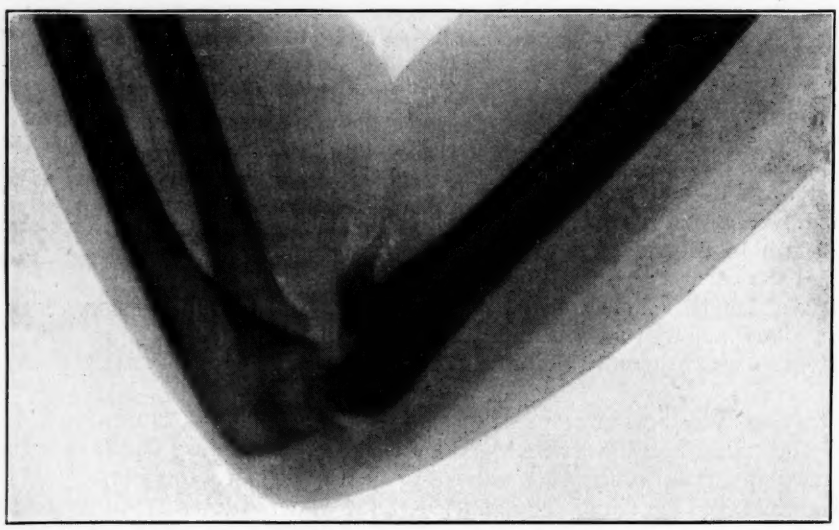

FIG. 378.—Skiagram taken on Aug. 30.

When last examined, in April 1922, ten months after the injury, the arm was practically normal. Movement through the full range was free and controlled, and the *x*-ray (*Fig.* 380) shows the further decrease in the new bone formation.

This case presents no extraordinary features, but it is of interest as showing the way in which the new bone becomes absorbed and the joint mobility restored when the

FIG. 379.—Skiagram taken on Nov. 16.

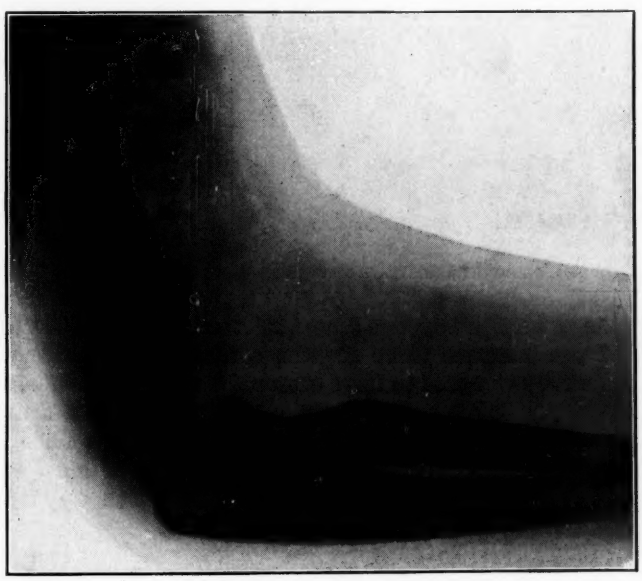

FIG. 380.—Final skiagram, April 2, 1922.

parts are put at rest. A consideration of the series of skiagrams may be of service when we are called upon to give a prognosis in a similar case.

2. Volkmann's Contracture.

The second complication is Volkmann's contracture. This calamity is fortunately rare, and in many cases preventable, but it is not so in all. As is well known, it is usually associated with the pressure of tight bandages and splints. Cases are reported, however, in which the condition has ensued on accidents when no splints or bandages have been applied. A full account of the condition and its etiology, together with references to the literature up to that date, is given by Dudgeon[2]. More recently an experimental study of the subject has been made by B. Brooks,[3] who summarizes his conclusions as follows: "That the classic picture of Volkmann's ischæmic paralysis could only be explained on the basis of acute venous obstruction would seem quite clear".

Suffice it to say here that the condition is reported to have followed the use of an Esmarch bandage, rupture or contusion of the main vessel of the limb, and thrombosis of the axillary artery.

Volkmann, in his article published in 1875, considers that the paralysis and contracture are caused by a cutting off of the blood-supply, and that the muscle dies.

Fig. 381.—From photograph showing the Volkmann's contracture.

The microscopical examination of portions of forearm muscle from the case about to be reported, as well as from similar cases, upholds this contention. The microscopic drawings figured illustrate this point particularly well when compared with those taken from a case of degeneration following division of a motor nerve. The condition is essentially different from that of degenerated muscle which has resulted from interference with the lower motor neurone.

In Volkmann's contracture there need be no interference with the peripheral nerves, and, in consequence, no sensory change. In point of fact the nerves are not infrequently involved, and it is therefore not uncommon to find an area of sensory loss.

In May, 1922, the patient, a boy, age 8, was referred to me. He had sustained a supracondylar fracture of the humerus three months earlier. The accident had happened in Jamaica, and the deformity was said to have been reduced under chloroform anæsthesia two hours later. The arm was bandaged with the elbow flexed. The arm swelled the same night, and the fingers were blue. The bandages were cut down after twenty-four hours, when the arm was blue and very swollen; blisters appeared next day, and the hand became contracted. Somewhat vigorous treatment had been applied, and I examined the boy for the first time three months after the injury.

He presented the appearance of a typical Volkmann's contracture (*Fig.* 381); the wrist-joint was flexed, the metacarpophalangeal joints were hyperextended, and the interphalangeal joints flexed. The deformity was fixed. The forearm muscles were hard, much wasted, and brawny; the elbow was practically fixed at a right angle, and a deep scar, the site of the original blister, crossed the external border of the forearm in the middle and upper thirds, and was down to, but not adherent to, bone. Anæsthesia was complete in the median and radial areas, incomplete in the ulnar. On electrical

examination the ulnar muscles were normal in reaction, but no faradic or galvanic response could be obtained from the median muscles.

The lesion was therefore diagnosed as Volkmann's contracture with complete physiological division of the median and radial nerves. The radial anæsthesia was obviously explained by the scar crossing the radial nerve, which was causing the interruption.

X-ray examination showed a supracondylar fracture of the humerus, with the usual gross displacement. The lower fragment was drawn upwards and displaced backwards, and the sharp lower end of the upper fragment projected into the antecubital fossa (*Fig.* 382).

It was decided, after a neurological consultation, that the median nerve should be explored. At the operation the nerve was found to be completely divided, with the usual end-bulb formation, and the division was obviously caused by the sharp projecting end of the upper fragment. It seems probable that this lesion, which is uncommon, was caused, not at the time of the accident, but rather at the subsequent manipulation. The forearm muscles were hard and yellow, and did not look like muscle at all. They formed an almost complete plaque of tissue, and it was not possible to separate the individual muscles. Suture of the nerve was effected without tension, and was worth while in view of the probable recovery of sensation in the hand. The lesion having occurred in the position in which the branches to the forearm muscles are given off precludes the possibility of their recovery, even if the state of the muscles had allowed of this.

FIG. 382.—Skiagram illustrating *Fig.* 381.

The further treatment of the patient was directed to a reduction of the deformity by splintage. The fingers could be extended when the wrist was fully flexed, and were held extended by a small splint made of plaster-of-Paris moulded to the hand, extending from the tips of the fingers to the wrist. A metal splint was then applied, taking purchase from the flexor aspect of the forearm above, and from the hand splint below. A felt pad was placed over the dorsum of the flexed wrist, and this was firmly bandaged to the metal splint. The result of the action of this force is gradually to extend the wrist, the fingers meanwhile being held extended by the hand splint. The splintage was maintained day and night for some three weeks, when the deformity was corrected.

Examination some months later revealed signs of commencing sensory recovery in the median nerve.

At the operation, portions of muscle, or what had been muscle, were removed. Dr. Greenfield cut sections of this, and we have had drawings prepared from typical sections, and, at the same time, drawings of a transverse section of normal muscle and of muscle degeneration following division of a peripheral nerve (*Figs.* 383–386).

The relative rapidity of the changes—the death of muscle in the ischæmic case taking place at the time—is manifest by a consideration of the sections, for, although there was a complete division of the median, the changes shown are not those of muscle degeneration due to peripheral nerve division, but of death of muscle and replacement by fibrous tissue.

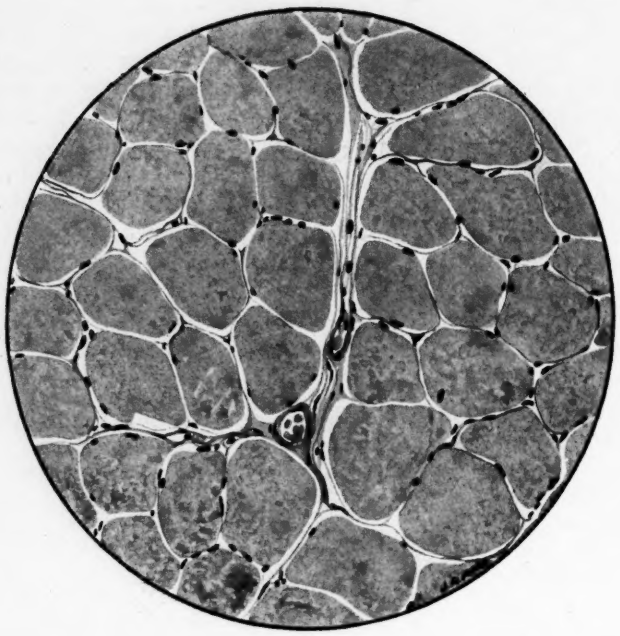

FIG. 383.—*Normal Muscle.* Transverse section, showing (*a*) the muscle bundles and their arrangement, (*b*) the sarcolemma nuclei, (*c*) the scanty fibrous tissue-stroma.

FIG. 384.—*Degeneration of Muscle* (after division of a peripheral nerve). Transverse section, showing (*a*) the muscle fibres shrunk, (*b*) the nuclei of the sarcolemma multiplied and, in some cases, passing into the fibre, (*c*) the thickening of connective-tissue septa. (In longitudinal section this muscle shows no cross-striation. This muscle is capable of recovery.)

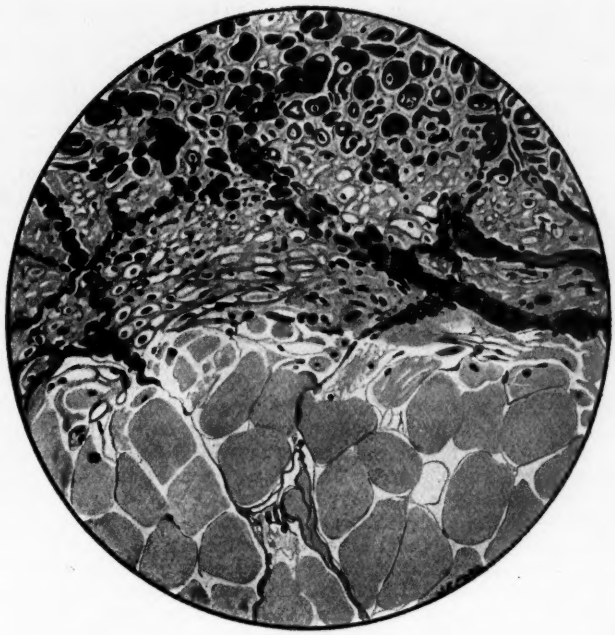

FIG. 385.—*Transverse Section of Muscle from the Patient*, showing (*a*) many oval fibres which do not fit in so well together as normally, (*b*) no sarcolemma nuclei, (*c*) a slight thickening of fibrous tissue and absorption by cells of connective tissue working in from the edge, (*d*) the very slight, if any, diminution in width of fibre. (The cells from the connective tissue have absorbed the muscle fibre and laid down connective tissue in its place. There is some muscle detritus left at the edge.)

FIG. 386.—*Longitudinal Section of Muscle from the Patient.* Passing from left to right across the section the following points are noticed: (*a*) dead muscle bundles in which cross-striation is very marked—actual fragmentation of the fibre in some sections at the cross-striation, (*b*) a few nuclei of cells spreading along the outside of the muscle fibres, breaking the muscle up into fragments, and absorbing the remains, (*c*) the fibrous tissue is laid down in regular bundles, replacing muscle bundles which have been removed by the phagocytic cells.

REPORT ON A PIECE OF MUSCLE FROM THE FLEXORS OF THE FOREARM.

The muscle was received fresh within an hour of the operation. One piece of it was fixed in formalin, another in trichloracetic-mercuric-chloride fixative (Heidenhain), and another in Zenker's fluid. The pieces fixed in formalin were treated by the Weigert-Pal method for myelinated nerve fibres, but none of these could be seen in any sections examined. The pieces of muscle fixed by the other methods were embedded in celloidin, and longitudinal and transverse sections were cut and stained by hæmatoxylin with van Gieson's counterstain. All showed a similar condition.

The most striking histological feature about the muscle was that over large areas no nuclei of any sort could be seen. This applied not only to the sarcolemma nuclei, which seemed to have disappeared completely everywhere, but also to the nuclei in the interstitial fibrous tissue, muscle spindles, and blood-vessels. At first sight one was inclined to blame the hæmatoxylin, as the differential staining of the picrofuchsin for muscle and fibrous tissue was well preserved. But where the sheath of the muscle or its tendinous attachment was included in the section, the nuclei in these structures stained perfectly. One had to conclude, therefore, that the nuclei had in reality completely disappeared from the muscle bundles and the other tissues contained in them. The muscle fibres, although they retained their normal arrangement, were shrunken and more rounded than normal. The cross-striation was extraordinarily coarse, and in some sections the fibres appeared to be fissuring across at the lines of the cross-striation.

The fibrous tissue between the muscle bundles was slightly thickened, but scarcely enough to constitute an abnormality. At the edges of the muscle where it was surrounded by its sheath, and at its junction with its tendon, there was everywhere a collection of round cells which lay between the muscular and fibrous tissue. These cells formed an almost continuous ring, usually not more than one or two cells deep, around the outside of the muscle. In some places they could be seen penetrating between the muscle fibres for a short distance from its outer border, but they never went deeper than about the third layer of muscle fibres.

Viewed under a higher magnification these round cells could be seen phagocyting the muscle fibres. All stages of this process could be seen. The cells at first were elongated or flattened, and lay closely applied to the muscle fibres. Then they became more rounded and passed gradually into the muscle fibre, which at the point of invasion lost its transverse striation and became granular. More cells then invaded the fibre, until over a considerable extent it was converted into a granular mass containing many cells, and only showing here and there the remains of its transverse striation. Then fibroblasts appeared, and fibrous tissue was laid down, at first in very thin threads and later more densely (*see Fig.* 386).

These processes led to a thickening of the fibrous tissue of the sheath, which, near the edge of the muscle, had a remarkable resemblance to muscular tissue. This appearance was best seen in transverse sections, which showed an arrangement of the connective-tissue fibres in rounded strands, often containing one or more nuclei, and in some cases showing inclusions of brownish granular material. The latter seemed to be the remains of muscle fibres which were undergoing absorption, and the appearance suggested that the fibrous-tissue cells were laying down fibroglia fibres in the position of the muscle fibres which had been absorbed (*see Fig.* 385).

The whole process seemed to be one of absorption and replacement of dead muscular tissue by fibrous tissue—the muscle acting as a non-septic, non-irritating foreign body, and being treated as such by the surrounding tissues.

It was impossible from the material at our disposal to say how far this process had gone, but apparently a considerable increase in the thickness of the sheath of the muscle had taken place. The process must necessarily be a slow one, as it was working only from outside the muscle and only to a very slight degree from the connective-tissue septa within the muscle. The latter, indeed, seemed to have died along with the muscle fibres, as they also contained no nuclei.

J. G. GREENFIELD.

The prognosis would seem to depend mainly upon the severity and extent of the original lesion, and in severe cases must always be bad. The deformity can be overcome by appropriate measures, but a return of voluntary power is not to be expected. Any power that is present will, under treatment, be conserved and will improve; but total inability voluntarily to flex the fingers to even the smallest degree, weeks or months after the onset of the contracture, is a very grave prognostic sign. The most that can be hoped for in such a case is to correct the deformity, which should always be possible, and possibly to restore sensation in an anæsthetic area.

In conclusion, I would express my indebtedness to Dr. Greenfield, who prepared and cut the sections, and who has written a note on the pathological findings.

REFERENCES.

[1] *Contributions to Surgery and Medicine*, Part iii, published 1887. London: H. K. Lewis.
[2] *Lancet*, 1902, Jan. 11, 78.
[3] *Archives of Surgery*, 1922, July, v, 188.

LENGTHENING OF THE TENDO ACHILLIS.

By H. H. GREENWOOD, Swindon.

The achievement of this operation by simple transverse section of the tendon is gradually falling into deserved disfavour. The gap caused by the wide separation of the severed ends of the tendon can only be bridged by fibrous tissue, with the not-surprising result that there is left undue weakening of this powerful tendon, in many cases necessitating an operation for repair of the defect.

Hemisection of the tendon at two points distant from each other one inch or more was a definite improvement, but still the operation was done subcutaneously by a tenotome. It was believed that the resulting fracture of the tendon could be represented by *Fig.* 387.

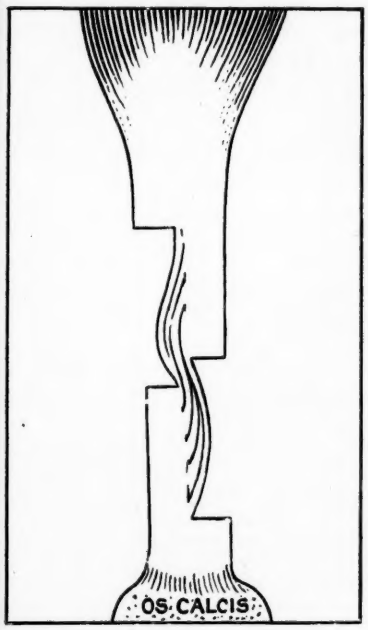

FIG. 387.—Hemisection of tendon, ideal result.

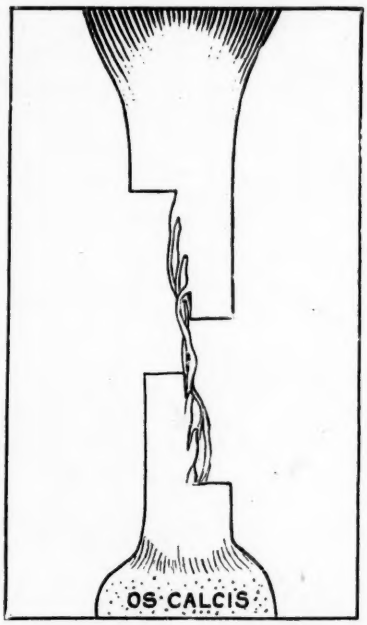

FIG. 388.—Hemisection of tendon, showing the snapping of one limb that frequently results.

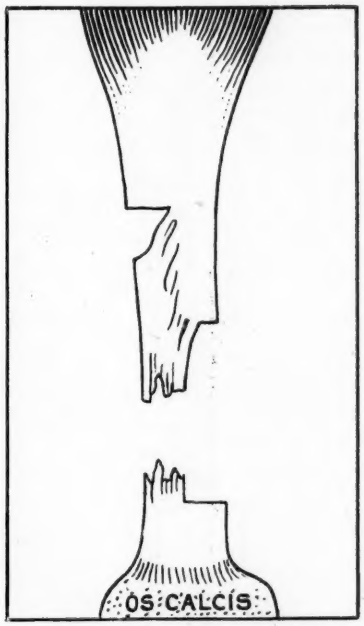

FIG. 389.—Hemisection of tendon, showing resultant wide separation of ends.

In reality the usual result is either snapping of one of the limbs of the incised tendon, as in *Fig.* 388, or wide separation of the ends, as in *Fig.* 389. Indeed, if the points of hemisection be more than one inch apart, the force required to produce a sliding tear is considerable, so much so that it is no matter for surprise that the condition shown in *Fig.* 388 is left. If we consider the mathematics of the question we shall see that much wider separation of the ends must occur than is imagined by those who still adhere to the closed operation. The undesirability of leaving a wide gap is realized by Calot,[1] for he says, "One ought to divide when it is merely a question of obtaining a lengthening of 1½ cm. in a child, or of 2½ cm. in an adult, because Nature may (not will) replace this amount of separation. But if you ought to obtain more than that you will perform elongation of the tendon."

The reality of this wide separation between the divided ends is not merely idle speculation, for repeatedly, after doing the usual hemisection by a closed tenotomy, and rotating the foot into the desired position, the marked depression that appeared on the site of section has impelled me to proceed to make a longitudinal incision and so put the matter to the proof. Invariably after exposing the divided ends I have found the condition depicted in *Fig.* 388 or *Fig.* 389.

Other surgeons have been driven by like considerations to adopt the open operation, deliberately lengthening the tendon to the extent demanded by the condition of the foot, and only exceptionally to do a closed tenotomy.

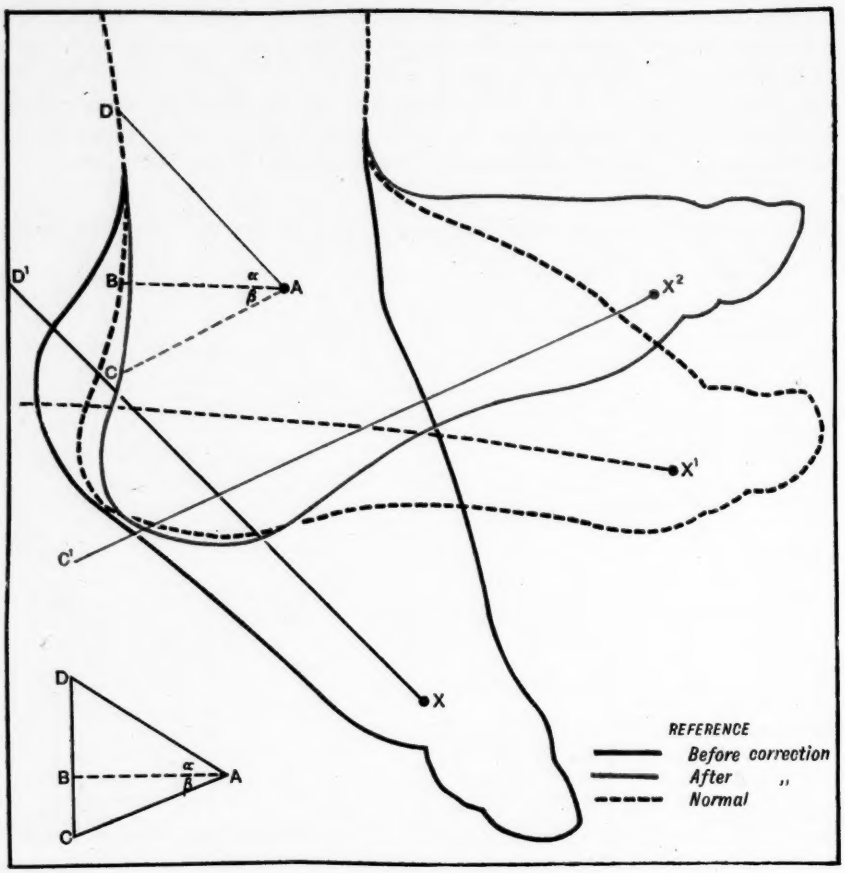

FIG. 390.—Diagram of foot illustrating method of estimating the required amount of lengthening of the tendon.

Mr. Fairbank[2] in an article on the "Orthopædic Treatment of Poliomyelitis," writes, "I prefer the open method . . . it is more accurate, and the ends of the tendon can be sutured so as just to allow the foot . . ."

No further justification for the open operation seems necessary, but there remains the question, hitherto unanswered, "By what means is it possible to estimate beforehand the extent to which the tendon in any given case requires lengthening?" An adequate reply will certainly lead to greater precision, and this I have attempted to furnish.

The ankle-joint allows a hinge-like movement round a horizontal coronal axis, which passes through the centre of the internal malleolus; this point lies further forward than is commonly depicted or appreciated.

In an investigation carried out by taking tracings of the feet of 60 normal children

at ages from 1 to 12 years, the average distance of the axis from the posterior border of the tendo Achillis (AB in *Fig.* 390) was found to be :—

From 0 to 2 years (10 examples)	..	$1\frac{1}{2}$ in.
„ 2 „ 4 „ „	..	$1\frac{7}{16}$ „
„ 4 „ 6 „ „	..	$1\frac{1}{2}$ „
„ 6 „ 8 „ „	..	$1\frac{3}{8}$ „
„ 8 „ 10 „ „	..	$1\frac{2}{3}$ „
„ 10 „ 12 „ „	..	2 „
In 10 adults (5 male, 5 female)	..	$2\frac{1}{4}$ „

A foot in a condition of talipes equinus lies at an angle which departs from the normal right angle to a variable extent—a very usual one is 45°. To obtain the best result, the foot should be moved round the axis of the ankle-joint until it passes the right angle to the extent of some 30° ; it is thereby made to traverse the full range of movement possible to a normal foot. It will be necessary therefore to move the deformed foot through an angle of 75° altogether.

In carrying out this movement, a given point D (*Fig.* 390) on the tendo Achillis will move downwards along the arc of a circle whose centre is the axis of the ankle-joint and whose radius is AD, to a point C. For practical purposes we may take the arc of this small segment as a straight line DBC. The line AD, meeting the posterior margin of the tendo Achillis at D, is drawn parallel to the longitudinal axis of the foot (which passes through the metatarsophalangeal joint X). The angles α and β are known, AD, AB, AC are approximately equal ; AD can be measured, AB can be taken from the average in the above table, and the distance DC (the extent to which we desire to lengthen the tendon) can be computed with sufficient accuracy for our purpose.

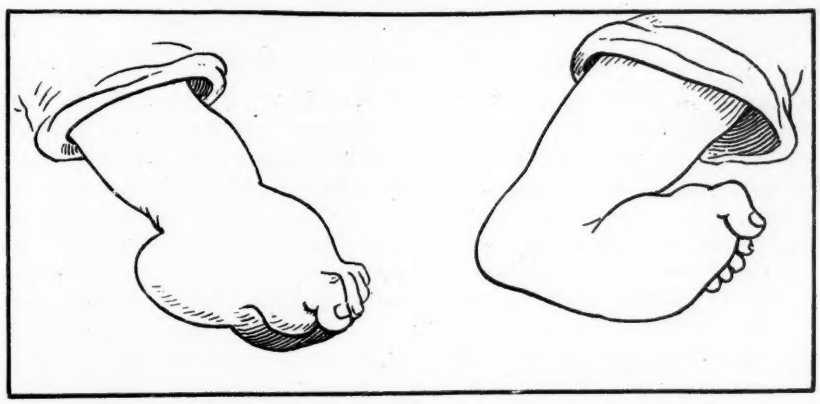

FIG. 391.—Talipes equinus in a child of $1\frac{1}{2}$ years, before and after operation.

For the sake of argument suppose the angle BAD = 45°, and BAC = 30°. We know that the average measurement of the line AB in a child of 5 years is $1\frac{1}{2}$ in., then

DC = DB + BC. But DB = AB tan α, and BC = AB tan β. Then

DC = AB tan α + AB tan β. But tan α (45°) = 1·0.
And tan β (30°) = 0·5774.

That is, DC = $1\frac{1}{2}$ in. × 1 + $1\frac{1}{2}$ in. × 0·57 = (roughly) $1\frac{1}{2}$ in. + $\frac{3}{4}$ in. = $2\frac{1}{4}$ in.

It will be seen, then, that in a child of 5 years a gap of $2\frac{1}{4}$ in. must be allowed for, and if it be desired that the ends should slightly overlap before suture, quite $2\frac{1}{2}$ in. For an adult in whom AB = $2\frac{1}{4}$ in. the gap will be about $3\frac{3}{8}$ in.

If it be objected that 45° for the angle DAB is extreme, and we take an angle of 30°, then for a child of 5 years the gap will need to be $1\frac{1}{2}$ in.

That these figures are substantially accurate I have confirmed repeatedly by actual measurements during the course of an open operation.

The angles chosen as examples represent those of a case of moderately severe talipes equinus, but the angle α sometimes approaches 60°. Reference to *Fig.* 391, which is a

drawing from a not uncommon type of case in a child of 1½ years, will support my contention that the angles assumed for the sake of argument are not excessive. The positions shown are before and after operation.

In the table subjoined I have worked out approximately the length of the gap that will be left in the case of three common angles at which the foot is found. The angle α varies, but the angle β remains constant. Given the premisses, it will be easy to compute these distances with greater accuracy if it is found desirable.

TABLE OF APPROXIMATE GAPS BASED ON AVERAGE MEASUREMENTS.

AGE	AVERAGE MEASUREMENT OF AB	LENGTH OF GAP		
		Angle DAB = 45°	Angle DAB = 30°	Angle DAB = 15°
0 to 2 years	1⅓ in.	2 in.	1⅓ in.	1 in.
2 „ 4 „	1 7/16 „	2 3/16 „	1 7/16 „	1 1/16 „
4 „ 6 „	1½ „	2¼ „	1½ „	1⅜ „
6 „ 8 „	1⅝ „	2⅝ „	1⅝ „	1⅛ „
8 „ 10 „	1⅔ „	2⅓ „	1⅔ „	1¼ „
10 „ 12 „	2 „	3 „	2 „	1½ „
Adult	2¼ „	3⅜ „	2¼ „	1 11/16 „

Tan 15° = 0·2679 Tan 45° = 1·00 Tan 75° = 3·7321
Tan 30° = 0·5774 Tan 60° = 1·7321 Tan 90° = ∞

It is difficult to appraise with exactitude the results obtained by the older methods, since they certainly do allow correction of the deformity to be achieved. Their chief defect lies in an unnecessary weakening of a limb that is already, as a rule, lacking in power. An open operation deliberately planned on the lines advocated above provides the enfeebled calf muscles with a tendon that is as nearly strong as the untouched one as it is possible to make it.

In doing the open operation, too, it cannot escape notice that there is usually brisk hæmorrhage, easily arrested and therefore of small moment; but in a closed tenotomy a considerable effusion of blood must often occur, hidden, but fated to organize and to leave unnecessary stiffness of the joint.

My own improved results, since adopting the open method for all but the mildest cases, leave me in no doubt as to the wisdom of this course. I will only add that a slightly curved incision, lying to the outer side of the tendon, gives the best exposure and causes the least hæmorrhage. A light plaster-of-Paris case applied immediately over a bandage made by cutting strips from common draper's wool is desirable. A continuous 'blanket' suture of 0 catgut gives good approximation of the skin edges, and can be left untouched until the plaster is removed.

REFERENCES.

[1] CALOT, *Indispensable Orthopædics*, 2nd ed., 678.
[2] FAIRBANK, *Brit. Med. Jour.*, 1921, April 9, 517.

OSTEITIS FIBROSA.

By R. LAWFORD KNAGGS, London.

(*Being the Hunterian Lecture delivered at the Royal College of Surgeons on January* 29, 1923.)

Osteitis fibrosa is the name given to a disease of bone in which part of the osseous framework and its contained marrow are replaced by fibrous tissue. In this, ossification usually proceeds in a more or less scattered manner—in some cases to such an extent as almost to reconstitute the bone.

As the microscopic appearances have some resemblance to those seen in other conditions, it materially assists the diagnosis if the lesion is so gross that the fibrous state and texture can be recognized by the naked eye. The conception of the nature of the affection is somewhat hazy, and cases are to be found recorded under various titles.

Though its name implies 'inflammation', yet its inflammatory character is accepted dubiously, and with reservation. A short description of certain allied conditions of whose inflammatory origin there is no question will, therefore, be a useful introduction to the main subject.

OSTEITIS FIBROSA ARISING BY EXTENSION FROM A JOINT AFFECTION, OR AS A RESULT OF SEPTIC IRRITATION.

In 1883 Arbuthnot Lane exhibited, at a meeting of the Pathological Society, the heads of both the femurs of a man, age 50, who had suffered from rheumatic arthritis of the hips. In the vertical section a mass of fibrous tissue was seen to extend inwards from the ligamentum teres and to blend in places with the under surface of the articular cartilage, which showed fibrillation at the point of contact. The changes were symmetrical.[1] Similar fibrous patches in the vicinity of rheumatic arthritic joints are described by Ziegler,[2] and the same author has depicted a cyst in a fibrous area surrounded by bony trabeculæ, in the same disease.[3] Again, Strangeways has pointed out that skiagrams of rheumatic arthritic joints will sometimes reveal transparent areas in the bones entering into their formation. These are found to be erosions of bone, or cavities in its interior filled with a gelatinous mucoid substance.[4]

The way in which such cysts originate has been described by Nicholson. When they form in the articular cartilage it is by liquefaction of the matrix, the disappearance of the corpuscles, and the formation of a fibrocartilaginous wall. When they occur in bone a corresponding degenerative change takes place. The bone trabeculæ in a limited area stain very slightly with eosin, and all traces of bone corpuscles and lamination are lost. These trabeculæ undergo resorption by large numbers of giant cells. The bone surrounding such necrotic areas is healthy, and the marrow fibrous, contrasting with the fat-marrow elsewhere. This fibrous marrow zone contains many leucocytes, but no giant cells. We may therefore presume that the mucoid contents of such a space are the result of mucinoid degeneration of the intertrabecular tissue and the vanishing bone.[5]

Analogous fibrotic changes may also originate from septic irritation.

In St. Bartholomew's Hospital Museum is a superior maxilla whose alveolar process, greatly increased in size, is transformed into a dense bony mass which reaches to the floor of the antrum, though that cavity has not been affected. Small scattered patches of fibrous tissue can be seen on the face of the section through the altered bone. The specimen (400*b*) was removed from a woman, age 38, who had noticed the swelling for twelve months. Some decayed teeth had been extracted three weeks before, and the

largest fibrous patch surrounds the alveolus which one of them had occupied. The microscopic structure was that of dense cancellous bone with its spaces filled by fibrous tissues showing some signs of mucinoid degeneration.

Thus we see that a fibrous osteitis may be an adjacent complication of different forms of inflammation, and that in some cases localized areas of degeneration may end in cystic spaces with fibrous boundaries, instead of patches of fibrous tissue.

OSTEITIS FIBROSA AS A PRIMARY AFFECTION.

Whilst it is clear that fibrous osteitis of a secondary character may be definitely associated with inflammatory lesions, it must be admitted that the precise nature of the changes in *primary osteitis fibrosa* is not so evident. The latter variety is a distinct clinical entity, is very liable to be confounded with osteomalacia, osteitis deformans, or with central bone tumours, and is very puzzling to the practitioner.

Its recognition is due to von Recklinghausen, who described and illustrated cases of it,[6] and Bloodgood[7] and Elmslie[8] have added to our knowledge. Bloodgood dealt primarily with bone cysts, but he incidentally defined various forms of osteitis fibrosa. His classification is instructive, and as I shall have occasion to refer to it subsequently, its principal groups may be quoted. He describes :—

1. Single cysts in which there is no connective tissue lining the cavity. It is always possible to find in such cysts a new connective tissue between the bone lamellæ of the shell (osteitis fibrosa).

2. Cysts with definite connective-tissue lining, which can be peeled off from the bony shell, and is identical, microscopically, with the fibrous tissue in the bone shell of the first group.

3. A small cyst or cysts in a solid mass of fibroid tissue. The medullary cavity is filled with the same kind of fibrous tissue as that in groups 1 and 2.

4. No cysts, but the bone shell filled with a solid mass of fibrous tissue.

5. Multilocular cysts. The distended shell of the bone is partitioned into multiple cavities which contain either fluid or fibrous tissue.

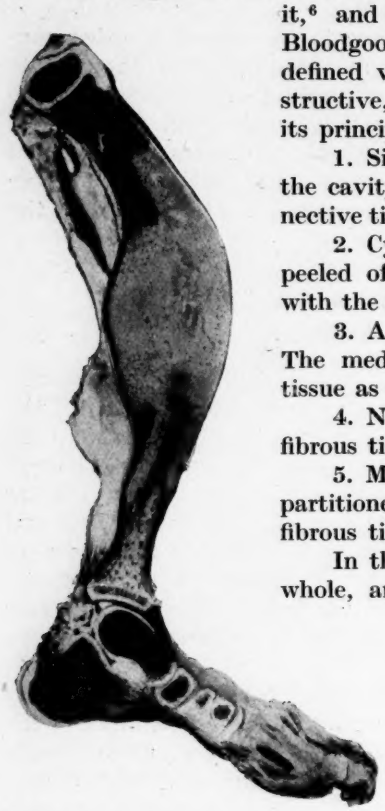

FIG. 392.—Tibia and fibula. (*Bilton Pollard's Case. University College Hospital Museum.*) (Reproduced from the BRIT. JOUR. SURG., 1914, ii, 53.)

In this paper, however, the disease has to be studied as a whole, and in proper perspective. The clinical cases, many of them classical ones,* which will be utilized for this purpose, fall naturally into one or other of the following groups :—

I. *Those in which the lesion is represented by a uniform mass of fibrous tissue.*

II. *Those in which a solid mass is showing signs of degenerating into one or more cysts.*

III. *Cases in which much bone is developed, and the disease shows some signs of coming to an end.*

IV. *Single cysts of bone.*

Group I.—CASES IN WHICH THE DISEASE IS REPRESENTED BY A UNIFORM MASS OF FIBROUS TISSUE.

Case 1.—The specimen in Bilton Pollard's case was removed from a child, age 5, who injured her leg when she was a year old (*Fig.* 392). A year and a half later the bone was noticed to be swelling, but was not painful.

* Several of these cases were recorded under other titles before osteitis fibrosa was fully established as a definite disease.

A solid mass of fibrous-looking material occupies the middle third of a bisected tibia, and sends extensions towards both cancellous ends. The bone is much enlarged in its middle portion, and bent like a bow. A small enlargement presenting similar characters exists in the fibula.

Sections showed anastomosing bone trabeculæ enclosing spaces filled with a material resembling the fibrillar matrix of growing bone. (Sp. 1341D, R.C.S. Museum.)[9, 10]

Case 2.—Bloodgood gives a beautiful photograph of a solid mass of fibrous tissue which Kammerer, of New York, curetted from the femur of a man, age 20. The skiagram showed it to have occupied the shaft in the vicinity of an old healed fracture.[11]

Case 3.—Elmslie records the case of a girl, age 18, in whom he curetted a cyst in the neck of a bent and shortened femur, and three weeks later removed a mass of firm fibrous tissue from the shaft.[12]

When the disease has reached this stage, the bone in section shows a solid area of fibrous tissue sharply differentiated apparently from healthy bone. But an earlier stage no doubt exists when the original bone structure is in process of disappearance. Of this stage we have but little knowledge, because, owing to the insidious and chronic character of the disease, the condition is not recognized until it has produced some deformity or led to fracture.

FIG. 393.—*Eve's case.* To illustrate the association of cysts with osteitis fibrosa. (*Spec.* 1968, *R.C.S. Museum, Gen. Pathol. Section.*)

Group II.—CASES IN WHICH A SOLID FIBROUS MASS SHOWS A TENDENCY TO DEGENERATE AND FORM CYSTS.

Case 4.—The most remarkable example of the association of cysts with osteitis fibrosa is the specimen presented to the College of Surgeons by W. T. Clegg, and investigated by Eve (*Fig.* 393). Originally thought to be a sarcoma, it is now catalogued as a soft fibroma ; but when osteitis fibrosa began to excite attention, Sir Frederick Eve, I believe, accepted that diagnosis. It was removed from a man, age 24, who ten years before had sustained a fracture of the tibia near its middle. A lump remained at the seat of fracture, and the leg always ached. Nine months before operation the leg began to swell and give pain, but the man walked until admission.

The preparations (R.C.S. Museum 1968, 1 and 2, Gen. Path. Series) are the two halves of an enormously expanded tibia—the one a macerated and the other a wet specimen.

The former is illustrated in Bland-Sutton's *Tumours* (*Fig.* 54, p. 94, 6th edit.). The latter shows the expanded tibia filled from end to end with a fibrocystic mass. The whole of its upper half is converted into four or five large cysts ; the lower half, with some cysts, is for the most part solid and of somewhat homogeneous appearance, though in many places a fibrous structure is apparent. All that is left of the bone is a reticulated shell and a small area of cancellous tissue beneath each articular cartilage. There is no sign of a capsule, and the edge of the solid tissue is at places definitely irregular, whilst delicate streamers from the mass can be recognized penetrating the bony shell at the side.

Under the microscope the solid part is seen to be composed of well-developed fibrous

tissue which in places is showing signs of degeneration. The cysts contained yellowish turbid fluid rich in cholesterin.[13]

Case 5.—A femur showing a small cyst developed in a large fibrous inset is pictured by von Recklinghausen in his monograph[14] (*Fig.* 394). The case from which it was taken had other features that make it worth while to record it, and I am indebted to Elmslie's paper for the following abstract :—

"The skeleton of a woman, age 66, who died of pneumonia. There was general hyperostosis of the skeleton, with cyst formation, enormous hyperostosis of the skull, hyperostosis and bending of the femora and the right humerus, and porosity of other bones, e.g., of the ribs. The bending had not arisen from previous fractures. In the bone-marrow there were patches of bone of ivory hardness, patches of spongy bone, islands of fibrocartilage, marrow tissue, and large cysts."

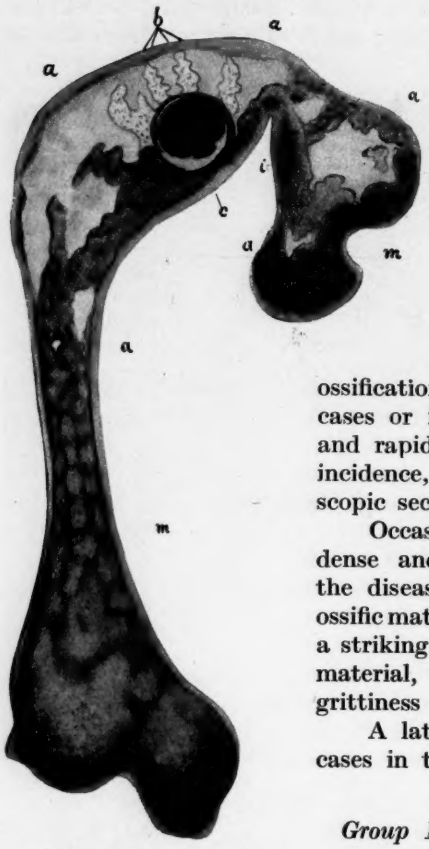

FIG. 394.—Femur from von Recklinghausen's case. (*a*) White fibrous tissue; (*b*) White porous bone formed within the fibroma; (*c*) Cyst with dark brown slimy material; (*m*) Several brown pigmented spots strewn in the yellow marrow and the remains of the spongiosa of the epiphysis; (*i*) Region of impaction. (Reproduced from the BRIT. JOUR. SURG., 1914, ii, 59.)

Case 6.—A femur very similar to the above is shown by Kuster from a female, age 17, and in addition to small cysts there is a circumscribed lobulated mass of cartilage occupying the neck. The last point is of some interest in connection with cyst formation in these cases.[15]

It would seem to be the usual thing for ossification to be going on in the fibrous areas except in those cases or in those areas in which there is evidence of early and rapid degeneration (*Case* 4). It is very variable in its incidence, the ossific points being numerous in some microscopic sections, and very sparse or absent in others.

Occasionally definite masses of new bone, sometimes dense and sclerosed, and evidently marking older patches of the disease, can be seen, but as a rule the production of ossific material in *Groups I and II* is not sufficient to produce a striking change in the naked-eye appearance of the fibrous material, though it is often sufficient to cause a feeling of grittiness when the flat side of a knife is drawn over it.

A later stage of the disease is to be recognized in the cases in the next group.

Group III.—Cases in which Much Bone is Developed and the Disease shows Signs of Coming to an End.

In two of the three cases that follow, the disease had been in progress for many years, and in the third it developed in adult life—a very unusual thing, as the onset is nearly always in childhood, or at least whilst growth is still going on.

Case 7.—This was recorded by Shattock and Bernard Pitts in the *Transactions of the Pathological Society*.[16]

A woman, age 37, after an injury when she was 31, developed a painful tibia which eventually led to amputation. The tibia in its upper half was transformed into a minutely cancellous bone-like tissue so devoid of lime salts that the bone was as pliable as india-rubber, and was cut readily with a knife. The compact wall and medulla were replaced by this tissue, and the medullary cavity was filled. The lower limit was abrupt, and the bone below normal. Under the microscope the soft tissue in the bone proved to be a highly cellular connective tissue, and the bone trabeculæ osteoid in character, their central portions being surrounded with a zone of acalcified matrix. (St. Thomas's Hospital Museum, 411 B.) (*Fig.* 395.)

Case 8.—This was the case of a man, age 53, in whom, during a period of great privation in early boyhood, the first sign of the disease appeared in the tibia. The limb was amputated for sarcoma, which had led to spontaneous fracture of the femur. The abdominal glands were affected when the patient was last seen, and a fatal termination was evidently imminent.

FIG. 395.—The tibia from *Case* 7. After Shattock and Bernard Pitts. (*Spec.* 411 *B*, *St. Thomas's Hosp. Museum.*)

FIG. 396.—Microscopic section from the lower end of tibia, *Case* 8. The fibrous tissue filling in the spaces between trabeculæ of new formation, and the absence of fat in the fibrous area, are well shown. Note the sharp demarcation of the fibrous area from fat marrow, and the long atrophic original trabeculæ which mark the separation. Low power.

The femur, the tibia, and the fibula were all extensively affected with osteitis fibrosa.

In addition to the gritty character of the fibrous tissue which replaced considerable portions of the cancellous structure of all three bones, masses or areas of very dense bone were present. These evidently represented the final stage of the disease. Some occupied the central medullary cavities, and had clearly been preceded by fibrous tissue.[17] (*Figs.* 396, 397.)

In these two cases (Nos. 7 and 8), beyond slight bending there was very little alteration in the shape of the bones.

FIG. 397.—A high-power view from the same microscopic slide. Note the non-laminated trabeculæ and the giant cells.

Case 9.—Frankling and Edgecombe's case. The patient was a lady, age 65, who gave a history of a deformed tibia from the time she was two years old. Previous to

amputation she had been under observation for more than two years, and a swelling over the upper part of the bone appeared about the beginning of that time. It gave the egg-shell crackling sensation, and it was feared that malignant disease had supervened. The tibia was the only bone involved.

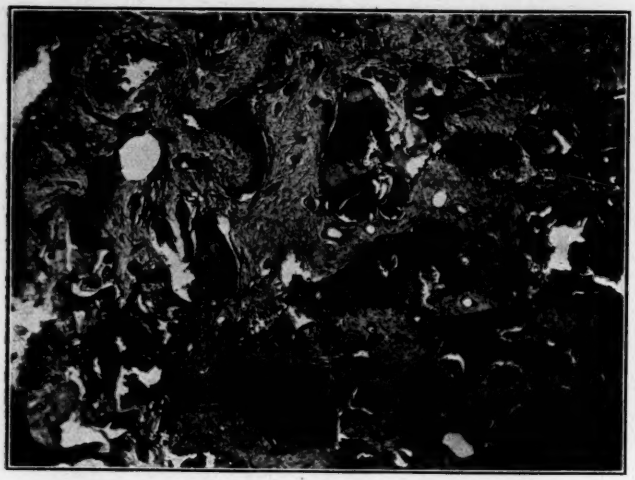

FIG. 398—Section from the chief fibrous mass in *Case* 9. (*Spec. R.C.S. Museum*, 711 *W.*) It shows the bone formation taking place from the fibrous tissue by metaplasia. × 30.

At the first glance the specimen (*see Fig.* 404) suggests a bone affected with osteitis deformans, owing to the bent shape, the uniform enlargement of the shaft, and the curious arrangement of bone in its interior. But there are at least three considerable masses of fibrous tissue present, and the largest, $2\frac{1}{2}$ in. long, occupies almost the whole sectional transverse area of the bone, reaching to the periosteum on the sides and in front. At its lower end it blends with a strong irregular fretwork of bone occupying the marrow cavity and gradually passing into the cancellous tissue of the lower end. (*Figs.* 398, 399.)

The other two masses of fibrous tissue lie amongst this bony fretwork, the spaces of which elsewhere are filled with fat. The latter is clearly a reversion from the connective tissue in which the new bone formation developed. The fat-marrow in the locular spaces is separated from the dense osseous septa by a fibrous membrane that can be lifted off the bone, which is seen to be dense and smooth and hard. It is probable that the bone in childhood would have presented an appearance very similar to that seen in Bilton Pollard's case (*Case* 1). (Half the specimen is in the Pathological Museum of the Leeds University, and the other half in that of the Royal College of Surgeons, No. 711 W.)

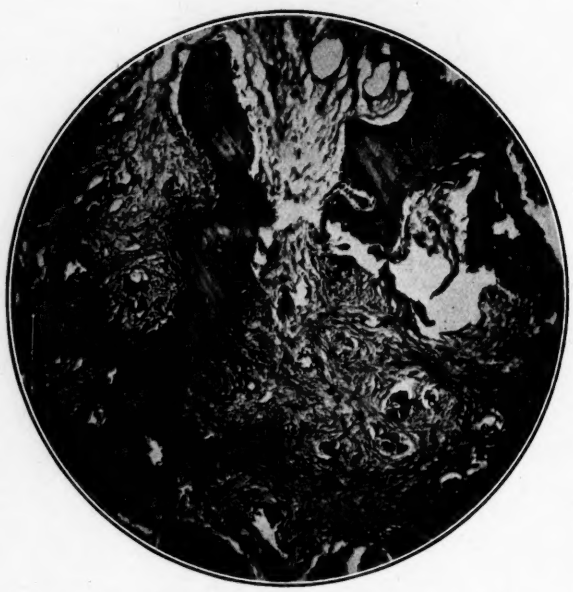

FIG. 399.—A portion from the same microscopic section as *Fig.* 398. × 55. (By Dr. G. H. Rodman.)

Group IV.—SINGLE CYSTS OF BONE.

Attention has already been directed to cases of fibrous osteitis in which a considerable mass of fibrous tissue contains one or more cysts (*Group II*). As a rule the cyst is only a minor matter in comparison with the amount of fibrous tissue in which it forms. There are cases, however, in which a cyst appears to constitute the whole pathological condition. Such cases fall into Bloodgood's first and second categories, viz., those cysts in which there is no lining membrane at all, and those in which there is a definite lining membrane

composed of condensed connective tissue. Yet even in the first group there is a zone of new connective tissue between the trabeculæ of the bony wall.

Is there sufficient justification for believing that these cysts originate in a pre-existing area of fibrous osteitis?

Apart from parasitic cysts, and degeneration or hæmorrhagic cysts in growths, cystic formation in bone may result from: (*a*) liquefaction of fibrous tissue (osteitis fibrosa); (*b*) liquefaction of cartilage (? chondroma, compare *Case* 6); (*c*) absorption of (necrotic) bone in such a manner as Nicholson has described as occurring in rheumatic arthritis; (*d*) the presence of a simple serous cyst (?)—at present this is hypothetical.

In the first three, a zone of young connective tissue forms at the periphery. We may suppose that it represents Nature's attempt to isolate the disease. The presence of such a zone would not by itself appear to justify the diagnosis of osteitis fibrosa cystica. That term should be reserved for those cysts that result from degeneration of the fibrous tissue which has replaced a portion of the bone structure.

Such an origin may be inferred when a cyst and a separate fibrous mass occur in the same bone (*Case* 3), or when a cyst occurs as part of the generalized disease; or when small patches of fibrous tissue are found in the immediate vicinity of the cyst.

Case 10.—Bland-Sutton's case of cyst in the humerus is an example of the last-mentioned variety, showing a small mass of fibrous tissue in the bone which intervenes between two portions of the cystic cavity, and establishes its real nature to the naked eye. (1637 D., R.C.S. Museum. Also *Fig.* 30, Elmslie's paper.[18])

Also the diagnosis might be considered proved by such a microscopic section as Bloodgood shows[19] in *Fig.* 23 in his paper (p. 161, Sower's case). It is taken from the bony wall of a cyst in the shaft of a humerus in which there was an unusually thick lining membrane. It is particularly instructive. Fibrous connective tissue is seen filling the spaces between the trabeculæ, and in it are numerous small cyst formations which suggest chat confluence of similar cysts is the explanation of the large one. *Case* 10 is typical of a form of osteitis fibrosa cystica which is probably not very rare. The majority of such cases occur in the upper ends of the humerus, femur, and tibia, and in the cancellous end of the diaphysis not far from the epiphyseal disc; but other bones and other situations are not exempt. The cyst is usually conspicuous in a skiagram.

Such cysts are liable to be brought to light when an injury results in a partial or complete fracture, and consequent disability. They contain a serous fluid which may be yellow and clear, or chocolate-coloured from old hæmorrhage, and in some cases cholesterin crystals are present. Cultures are usually sterile.

HISTOLOGY.

The histology of osteitis fibrosa is, in the main, fairly constant.

1. The ordinary bone-marrow is replaced by a dense vascular connective tissue.* This is composed of fusiform or branched cells with outrunning processes. It may assume the appearance of ordinary fibrous tissue and even show a whorled arrangement.

2. All the fat disappears.

3. The osseous framework has given place to this connective-tissue development. From the thoroughness of its removal it may be surmised that it vanishes with unusual rapidity, but there is very little evidence of the way in which it is destroyed.

4. Throughout this connective-tissue replacement numerous scattered foci of new bone are forming. These foci grow into trabeculæ, which in turn coalesce and form a network. Eventually they develop into sclerosed masses of bone, whose fibrous character is often very apparent under the microscope (*Fig.* 400).

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* A similar change in the marrow occurs in some other diseases. This is so in osteomalacia, in rickets, and in osteitis deformans; but each of these diseases has distinguishing histological features. In osteomalacia identical ossific foci in the connective tissue may *sometimes* be seen; but whilst new bone formation is the rule in osteitis fibrosa, it is rare in osteomalacia except in callus. Again, whilst the new bone trabeculæ in osteitis fibrosa are usually completely calcified and only occasionally assume the osteoid form, in osteomalacia they are always incompletely calcified and composed of osteoid tissue.

In a microscopical section from the fibrous area in a case of osteitis fibrosa, ossific points or small trabeculæ may be seen, often in considerable numbers, scattered irregularly through connective tissue. In some parts of the section they constitute its most conspicuous feature; in others they may be rare or altogether absent. The new trabeculæ are not laminated—certainly not in the early part of their growth—and the bone-cells are large, rounded or triangular, and not flattened and stellate as in normal bone. At the periphery of the disease such new trabeculæ may sometimes be seen based upon old laminated ones (*see Fig.* 396).

Ossification *begins either by metaplasia of small patches of connective tissue* (*Fig.* 401), or by deposit of calcareous granules round a connective-tissue cell in a matrix formed by the connective tissue itself. A group of a few adjacent cells, so altered, form a small calcareous mass, and the fibrils of the connective tissue can be traced into its sides. At its edge cells appear—as osteoblasts—in the spaces between these fibrils, and are gradually incorporated in the bone, which, as it grows, assumes the characters and form of a trabecula, but without the normal lamination. Growth and resorption are active in connection with these new formations, especially where the connective tissue is very cellular—a row of osteoblasts and several osteoclasts may often be seen applied to the edge of a single trabecula, and the modelling process is evidently going on energetically. (It may result in laminated trabeculæ.)

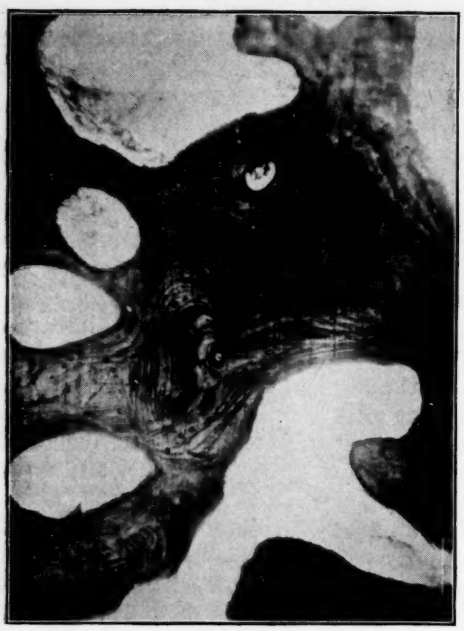

FIG. 400.—From the same case as *Figs.* 401, 402. Part of a patch of sclerosed bone in the fibrous mass. An under-exposed plate to show the architecture of the bone, but failing to produce the connective-tissue marrow, which fills the lacunar spaces.

An intermediary stage of fibrocartilage in the ossifying process has been observed (Elmslie), but is very uncommon; and the formation of osteoid trabeculæ, to which a reference has already been made, would seem to occur in cases in which a large amount of new bone has been laid down (*Cases* 7 and 8).

5. The origin of the cysts is not very clearly traceable in the microscopic sections. In certain cases there is a tendency for tracts of the connective tissue to pass into a state of necrosis, leaving only a framework of myxomatous tissue, the nuclei failing to stain. Such areas of degeneration are probably a first step to the production of a cyst, but they are met with not only where cysts have already formed, but in other long-standing cases in which no such tendency has shown itself. The cyst contents are in most cases a pale yellow serum, which suggests that the process of softening arises from a liquefactive rather than a mucinoid degeneration of the fibrous tissue. The section from Sower's case, previously mentioned, would seem to indicate that numerous minute cystic spaces first develop, and that a large cyst results from their coalescence.[19]

6. Finally, it should be clearly understood that there is a marked absence of the small-celled infiltration met with in the more acute forms of inflammation.

THE CLINICAL ASPECT.

The study of the different groups of cases enables us to follow not only the evolution of the affection, but also its clinical progress. The disease usually begins in childhood or during the growing period, and if left to itself may last a lifetime. There is some reason to believe that it may become stationary, or even go on to a spontaneous cure.

Three types may be distinguished :—

1. A limited local deposit, frequently becoming a cyst.
2. A more diffuse affection of a single bone involving the whole or a considerable part of the diaphysis.
3. A generalized form in which many bones are affected.

The patient only comes under observation when the disease is pronounced, and for one of the following conditions :—

1. An enlargement of the bone, not readily noticed unless the bone is comparatively superficial.
2. Deformity due to bending of the affected bone or bones.
3. Fracture, which may be partial or complete and often spontaneous.
4. A limp caused by shortening.

FIG. 401.—From the surface of the same section as *Figs.* 400, 402. Showing the formation of bone by metaplasia of the connective tissue. *Figs.* 400, 401, 402 are from different parts of the same microscopic section. (By Dr. O. C. Grüner.)

A fracture is a very common complication. Four cases, at different times, came under my observation at the Leeds Infirmary; three were of the generalized type, and all three had suffered from one or more fractures. One of these was a boy, age 12 (Gerald G.). At a time when both arms were in splints for fractures of each humerus, he was trying to raise a door latch with his head when both his femurs broke 'with a crack'. All the fractures united, and it fell to me to remove three myelomata from his upper and lower jaws. He died two years later of 'heart failure', and during the last ten months was bedridden.

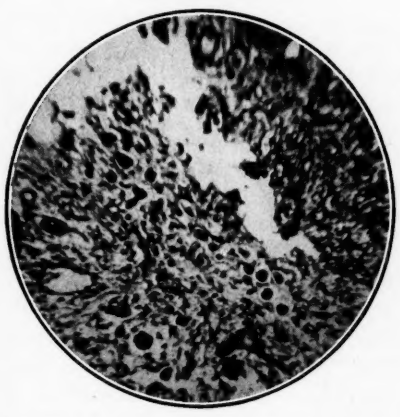

FIG. 402.—From Littlewood's case of osteitis fibrosa of lower jaw. A part where many giant cells were grouped together. (*Leeds Med. School Museum.*)

In some cases the disease is of such a mild character that a sufferer may be able to continue in active work throughout a fairly long life without suspecting that he is the subject of a progressive disease, until laid up by a late and probably final complication. In other cases a patient may welcome amputation to be rid of the incubus of a deformed and useless limb.

The long bones are most commonly attacked, but the skull is also frequently the seat of disease. Under the somewhat vague appellation of leontiasis ossium, Victor Horsley described five cases of hyperostosis of the frontal bone. Four of them were almost certainly examples of osteitis fibrosa.

My fourth hospital case, a girl, aged about 16, had a swelling on the frontal bone which was so small that its removal was accomplished by a trephine with a very large circle. This cut through the bone with the greatest ease. The histology was that of osteitis fibrosa.

The ossifying process that goes on in the fibrous tissue may be regarded as Nature's attempt to produce a cure, but it rarely comes to anything; for if the bone is made firm at one part, the disease is usually in active progress at another.

Fibrous osteitis is occasionally complicated by tumour formation. The common tumour is giant-celled myeloma. This might be expected, seeing that the disease involves the medulla and is often marked by considerable giant-cell development (*Fig.* 402). Malignant disease may also supervene and terminate life. An abnormal tissue in a state of ill-regulated activity for many years is an obvious predisposing cause of such a complication.

In *Case* 8 a spindle sarcoma caused spontaneous fracture. The connective tissue could be traced till it merged in the growth, and a nodule in the adjacent muscle was "composed of a similar spindle-celled growth supported in a connective-tissue basis identical with that seen throughout the three bones".

FIG. 403.—From a section taken from Howship's case of osteomalacia. (*Spec.* 739, *R.C.S. Museum.*) On the right is seen fat infiltrated with recently effused blood. The trabeculæ are completely decalcified except for a very small fragment (shaded deeply) in the lower part of the omega-shaped trabecula. The whole cortex is very thin, the periosteum lying immediately on the left of the two isolated trabeculæ on the left side and just failing to be included in the drawing. The breaking up of the decalcified substance and its metaplasia into fibrous tissue is well shown in the upper part of the drawing. (*Drawn with camera lucida by J. R. Ford.*) *See* footnote.

THE PATHOGENESIS.

The disease includes the removal of a tract of osseous tissue, and its replacement by fibrous or connective tissue in which ossification takes place in an attempt to repair the damage.

Why is the bone removed?—Possibly because some change has occurred in it which makes its removal necessary. The most probable change is an impairment of its vitality to such an extent as to cause its death or render it incapable of recovery.*

How can such a change in a bone's vitality be caused?

In the suppurative inflammations which end in necrosis, bacterial toxins play a considerable part. Not only do they excite the inflammation which kills the bone, but they exert a harmful influence upon the bone and its soft tissues before the blood-supply is cut off. Owing to the intensity and rapidity of the process, this latter influence is of very little moment.

In tuberculous inflammation, toxic influence no doubt prepares the way for the more ready disintegration of the bone trabeculæ by tuberculous granulation tissue. But in this instance it is more easy to appreciate the action of

* *How is the bone removed?* It is likely that removal is accomplished in one of two ways: (1) By the removal of necrotic trabeculæ by giant cells in the way described by Nicholson (*v. supra*); or (2) By some such process as that shown in the accompanying drawing (*Fig.* 403). This is from a case of osteomalacia (No. 739, R.C.S. Museum), and shows fragmentation and disappearance of osteoid tissue. It will be noticed that there are no giant cells taking part in the process.

the poison. In both these conditions the toxins are produced locally, and it is easy to infer their formation because the presence of micro-organisms can be demonstrated.

But osteitis fibrosa is clearly not dependent on a local micro-organismal growth. Nevertheless, by analogy, we may suspect that the preparatory changes in the bone which necessitate its removal are also caused by toxins. In that case the poison is carried to the part by the blood-stream.

The toxic substances may originate: (1) From micro-organisms; (2) From tissue metabolism (compare CO_2); or (3) From intestinal sources.

It is highly probable that a toxæmia capable of producing the damage may, in different cases, be derived from organisms of different kinds, or be of metabolic or intestinal origin. It is even possible that the source may vary at different times in the same individual. It is important that we should realize that toxins are not necessarily specific in the same sense as pathogenic micro-organisms. But, besides toxins, another factor comes into play, viz., *the vitality of the tissues, and their ability to resist toxic influence.* It is a matter of common knowledge that the power of resistance to infection by micro-organisms is possessed by different individuals in very different degrees, and that even families may show proclivities to certain forms of disease in consequence. But we may go a step further, and recognize that there is a tissue resistance to toxic influence, and that one organ or tissue in the individual may show it in less degree than the others. This again is well known. Such an organ or tissue constitutes a 'locus resistentiæ minoris', and its resistance may be broken down by toxins arising in different ways.

FIG. 404.—Skiagram of tibia from *Case* 9. Frankling and Edgecombe's case. A partial fracture is seen at the upper part. The pale area below is the main fibrous mass. Below that a fretwork of bone filled in with fat and occupying the central canal is indicated. Still lower other fibrous masses, above and below more indications of bony fretwork, can be recognized.

This may be illustrated by an example. A transient toxic nephritis (hæmaturia, etc.) was in the first instance excited by a septic infection, a second time by an influenzal infection, and a third time by an intestinal one.

The toxins may or may not have the same chemical composition, but they certainly have irritating properties, which may fail to injure the tissues as a whole, but may select and influence the one that has least power of resistance. Moreover, the usual circumstances that depress the vitality of tissues may cause such an one to succumb, though it may have resisted successfully till exposed to them. If this reasoning is correct, it follows that the onset of osteitis fibrosa may be explained on the supposition that, in certain individuals, an injurious influence is exerted upon the bones by toxins, which may be derived from one or more of various sources; that such toxins pick out the bones because they happen to be the tissue of least resisting power in the particular individual;

and that in many susceptible people they would fail to produce any obvious effect at all if some depressing influence (e.g., a fracture) had not lowered resistance still further, and determined the point of assault.

Little is known of the precise way in which the osseous tissue is affected by the toxins. The main mass of bone in a diseased area has disappeared before opportunities for microscopic investigation occur. But in trying to appreciate what takes place as a result of their action, we may picture to ourselves that both bone trabeculæ and marrow are affected, but that the trabeculæ, composed of a calcified matrix with only a few cells in its substance, are more vulnerable than the marrow. The influence that may damage the trabeculæ beyond the possibility of recovery may excite reaction in the marrow. The tissue that is produced by this reaction is probably active in removing the moribund trabeculæ, with or without the aid of giant cells. At the first stage of the process with which we are familiar there is found, substituted for the bone and its marrow, a uniform tract of connective tissue exceptionally well supplied with blood-vessels. This tissue is osteogenic, and in places the beginnings of a new bone-formation are to be seen. Subsequent progress consists in a steady advance of irregular ossification. Resorption accompanies it, but whether this process is simply adaptive and healthy, or a removal of parts of the new bone brought under the influence of a continuous supply of toxins, it is impossible to say.

At last, in parts of a diseased bone the natural termination of the morbid process may be reached. The new bone becomes dense and sclerosed—in some places forming masses, in others strong septa—and the remaining connective (osteogenic) tissue reverts to adipose medulla (*Fig.* 404).

These various changes point to an attempt on the part of the body to remove a portion of the framework of a bone which has been incapacitated beyond repair, and to replace it by a fresh development. When this has been accomplished, Nature demobilizes. The actual process by which the substitution is accomplished is, by most authorities, regarded as *inflammatory*, in spite of the fact that ordinary inflammatory cells are never in evidence. This view has been justified on the ground that " the new material has the loose connective-tissue structure of inflammatory new formation " (Report of the Committee of the Pathological Society on Morbid Growths : Messrs. Shattock and B. Pitt's case). If the interpretation of the nature of the morbid process which has been suggested is correct, the disease would certainly comply with the conditions laid down by Burdon Sanderson, viz., " Inflammation is the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality."

TREATMENT.

This should naturally concern itself in the first place with the cause. Any existing focus of sepsis should be removed. In a jaw case which I had the opportunity to investigate at a considerable interval after the removal of septic teeth, there was definite improvement and the hypertrophy had come to a standstill.

The dietary of these cases opens up a wide field for observation and experiment. There is good reason to believe that the pathogenesis of such diseases as rickets, osteomalacia, osteitis fibrosa, and osteitis deformans is allied. There is evidence that diet influences toxin production. It is not unlikely that Nature provides the antitoxin ready to our hand if we can only recognize and identify it. It matters not whether we call it ' antitoxin ' or ' vitamin '.

Tubby[20] records the case of a medical man who suffered from osteitis deformans. It is very significant. The patient attributed his improvement to the adoption of a diet rich in proteins and very sparing in carbohydrates; and the whole malady entirely to error in diet—viz., to lack of protein mainly, but partly to excess of starch. Eating potatoes never failed to produce a return of his pain.

Any measures that will assist in increasing the resisting powers of the patient, or prevent exposure to depressing influences, are of course indicated.

Various surgical procedures have been adopted in suitable cases. When a fibrous mass has been sufficiently local, its removal seems to have been justified by results. Bloodgood is of opinion that, if an osteotomy is required, curetting should be combined with it. Cysts have been curetted and packed, or filled in various ways, and the portion of bone affected by the disease has been excised and the gap dealt with. Hæmorrhage is the real danger in curetting operations when the disease is extensive, and fatal cases have been recorded. In some cases amputation may be advisable as an operation of expediency; a malignant complication of course renders it one of necessity.

OSTEITIS FIBROSA IN GOATS.

In the Pathological Society's Transactions (1889, vol. xl, p. 449) W. G. Spencer has given an account of a disease met with in goats. The affected animals were drawn from South London, where they had been kept for milking; consequently females predominated.

Before they were attacked by the disease they were fat, and their coats were smooth. Their diet had been hay, corn, cooked vegetables, and garbage, and they differed from goats fed on their natural food—hay and coarse upland grass—in that the latter are usually thin and have staring coats. Their ages varied from two months to three years.

The disease manifests itself by symmetrical swellings of the mandible, which gradually increase until the mouth cannot be closed, and death results from starvation, owing to the arrest of rumination in consequence of the immobility of the jaw. With the exception of changes in the upper and lower jaws, no others are perceptible, as a rule, during life.

The disease runs a rapid course, death supervening from one to two months after the jaw swellings have become evident. The enlargements are produced by a new formation of homogeneous appearance and firm elastic consistency, which replaces the original bone. They are most noticeable in the lower jaw, and involve particularly the angles and rami, gradually shading off in the body. The upper jaws may also suffer, but to a less extent.

FIG. 405.—Osteitis fibrosa in the upper jaw of a goat. (*Spec.* 715, *R.C.S. Museum.*) A mass of fibrous tissue is shown, embracing the roots of a tooth and another mass adjacent to the nasal fossa. Possibly they are portions of a single mass, appearing as separate ones owing to the point at which the section was made.

The swelling, when cut across, is solid throughout, of a pinkish-white colour, and spicules of bone can be felt to grate under the point of the knife. Both the skull and the long bones may also be affected, and become infiltrated by the new tissue. In one case the upper end of the tibia was replaced by the same pinkish-white growth as that in the jaws. Even enlargements of the bones may result, and they can easily be cut with a knife. After maceration the bone is extremely light, of the texture of fine sponge, and may fall to pieces during the process. There is no evidence of rickets, and the epiphyseal lines are healthy. The changes in the jaw are said to begin in the interior of the bone about the tooth sockets. An upper jaw in the Royal College of Surgeons Museum (715D) (*Fig.* 405) shows a patch of new formation embracing the roots of a healthy tooth, but this is separated by cancellous tissue from another patch. In specimen 366C in the St. Thomas's Hospital Museum, the bulk of that part of the swelling involving the body of the jaw intervenes between the erupted milk

teeth and the unerupted permanent ones, which are displaced downwards close to the lower border of the mandible—all the teeth are healthy.

There is no true capsule to the new tissue formed in this affection. The morbid material merges with undestroyed bone, or extends to the periosteum, or blends with the peripheral bony layer which intervenes between it and the periosteum. In the St. Thomas's specimen already referred to, numerous small cysts the size of a pin's head are scattered throughout the mass, and these are larger and more crowded in the immediate neighbourhood of the embedded permanent teeth. Similar cysts can be seen in other specimens. A macerated lower jaw (715F, Royal College of Surgeons Museum) shows the interior of the swelling to be largely filled with a spongy or finely granular and excessively friable osseous material, which easily separates in powder. Obviously an osseous trellis-work permeates the new formation at some period of its development. On the other hand, a sagittal section through the right femur of a goat, age 3 years (715H, Royal College of Surgeons Museum), shows only a thin compact shell with very slight remains of a rarefied cancellous tissue adhering to its inner surface. In the recent state it was filled with soft material.

Histologically the new formation (studied in the St. Thomas's Hospital specimen) consists of a matrix of close connective tissue in which lies a network of osteoid or imperfectly calcified trabeculæ. It is impossible to distinguish it from a section of fibrous osteitis in which the new bone formation is of an osteoid character, or from a section of callus formation in osteomalacia. There can be no doubt that the bone condition is osteitis fibrosa, and there is nothing to show that it has any connection with rickets. But the fact that in goats the jaws are specially selected for attack, though the teeth show no sign of disease, suggests that the act of rumination is probably a predisposing factor.

In its early stage the disease may be associated with a joint condition. Several joints may be affected and they contain darkly-stained synovial fluid. The synovial membrane is swollen and gelatinous, with dark hæmorrhages into it, and the cartilage may be eroded in spots and the bone exposed. The affection, however, is so slight that it may be overlooked, and the swelling which denotes it may disappear, leaving only slight traces behind. It is not clear how this peculiarity of the goat's disease is to be interpreted.

The author takes this opportunity to acknowledge how greatly he is indebted to Professor Shattock, not only for his assistance in placing much pathological material at his disposal, guiding him through the pitfalls of histological investigation, and directing him to reliable sources of information, but for much kindly criticism and other help.

He also desires to express his obligation to Dr. O. C. Gruner and Dr. G. H. Rodman, whose beautiful photomicrographs materially add to the value of this paper.

REFERENCES.

[1] *Trans. Pathol. Soc.*, xxxv., 299.
[2] Ziegler, *Pathologische Anat.*, 1906, ii, 279.
[3] *Virchow's Archiv.*, 1877, lxx, 502.
[4] *Bull. of the Committee for the Study of Special Diseases*, i, No. 6, 93.
[5] *Ibid.*, 1907, i, No. 7, 109.
[6] Von Recklinghausen, *Virchow's Festschrift, Assistenten*, 1891.
[7] Bloodgood, *Ann. of Surg.*, 1910, lii, 145.
[8] Elmslie, *Brit. Jour. Surg.*, 1914, ii, 17.
[9] *Trans. Pathol. Soc. London*, 1884–5, xxxvi, 388.
[10] Elmslie, *Brit. Jour. Surg.*, 1914, ii, *Fig.* 51.
[11] *Ann. of Surg.*, 1910, lii, 148.
[12] Elmslie, *Brit. Jour. Surg.*, 1914, ii, 38.
[13] Eve, *Trans. Pathol. Soc. London*, 1888, xxxix, 273.
[14] *Virchow's Festschrift, Assistenten.*
[15] *Arch. f. klin. Chir.* 1897, lv, 594.
[16] *Trans. Pathol. Soc. London*, xlvii, 177.
[17] Knaggs and Gruner, *Proc. Roy. Soc. Med.* (*Pathol. Sect.*), 1908, 26.
[18] Elmslie, *Brit. Jour. Surg.*, 1914, ii, 17.
[19] Bloodgood, *Ann. of Surg.*, 1910, lii, 161. *Fig.* 23.
[20] Tubby, *Deformities, including Diseases of Bone and Joints*, 1912, vol. ii, p. 569.

CHRONIC DUODENAL ILEUS.

By SEYMOUR BARLING, Birmingham.

The condition of acute gastroduodenal ileus, the so-called idiopathic dilatation of the stomach, which may arise as a post-operative complication after any surgical procedure, or even occasionally as a primary condition, is well recognized, but the closely related chronic condition has had little attention paid to it in this country till Wilkie's[1] paper focused attention on the matter. The condition is commoner than the literature on the subject would lead one to suppose, and both from the symptoms directly due to it, and from the part it plays in the production of other abdominal disorders, is worthy of further study.

Chronic duodenal obstruction may arise from a number of causes: some congenital in origin, such as partial atresia, ring pancreas, or excessive duodenal fixation by adhesions; in other cases neighbouring inflammations or growths may act by invading the duodenal wall or embarrassing its musculature, by kinking or direct pressure. In both acute and chronic ileus a most frequent cause is obstruction by the pressure of the mesentery and its contained vessels as it crosses the viscus. As the superior mesenteric artery and vein pass downwards across the third part of the duodenum, they normally produce a slight narrowing of the lumen at this point; in cases of visceroptosis or in abnormalities of the mesentery or the origins of these vessels, the duodenum may be excessively compressed near its termination.

In the adult the duodenojejunal junction is the point of transition of a relatively fixed portion of the alimentary canal into a mobile portion. Furthermore, the degree to which this process of fixation of the duodenum occurs in the embryo is one which varies within wide limits: it is especially towards the more distal parts of this length of bowel that variation occurs.[2] The fixation of the duodenum occurs primarily by fusion of the mesoduodenum with the mesocolon and the structures lying in the neighbourhood of the right kidney; in addition, other secondary adhesions occur, especially around its terminal part. The frequency and variation of the peritoneal pouches around the duodenojejunal junction are explained by the irregular occurrence of these adhesions. Again, the neighbourhood of the third part of the duodenum is the pivot around which intestinal rotation occurs, rotation which brings the cæcum across from the left to the right side of the body and carries the root of the mesentery of the jejunum and its contained superior mesenteric artery and vein athwart the duodenum near its junction with the jejunum. Embarrassments thus produced by abnormal adhesions and by the presence of peritoneal pouches into which herniation may occur, may be additional factors in rendering this part of the bowel vulnerable to obstruction by the crossing of the mesenteric vessels, themselves liable to great variation in tension, depending on the length of the mesentery, posture, and the presence or absence of distention of the small intestines.

Compression of the third part of the duodenum by the mesenteric vessels as a cause of acute gastric dilatation was suggested by Rokitansky[3] in 1849. In 1889 Glénard[4] regarded the dilated stomach as dragging on the duodenojejunal junction and so causing chronic obstruction at this site. Albrecht[5] reported cases of chronic obstruction in 1899 due to flattening of the duodenum beneath the mesenteric vessels, and demonstrated the obstruction by experiment. Robinson,[6] in 1900, pointed out the importance of mesenteric obstruction of the duodenum as a cause of gastroduodenal dilatation, and gave an account of the clinical symptoms and autopsy findings in chronic cases of this nature.

From this time onwards, with increasing opportunity of studying the relationship of the parts in the living, many papers have been devoted to the subject: notably by

Conner,[7] Bloodgood,[8] and Codman.[9] The last drew attention to the local and general toxic effects produced by duodenal stasis and its action as a causative factor in the production of chronic duodenal and gastric ulcer, cholelithiasis, and pancreatitis.

The studies of Wipple,[10] Sweet,[11] Ellis,[12] and many other workers as to the cause of death in acute intestinal obstruction and the production of toxic substances in the mucosa of the duodenum and jejunum under conditions of stasis, throw further light on the etiology of acute and chronic gastroduodenal ileus. The toxin is a systemic poison, causing the rapid collapse and death in the acute cases; whilst in the chronic ones its local action is related to the causation of chronic gastric and duodenal ulceration and gall-stones.

Kellogg[13] reviews the whole subject in 1921; he gives particulars of 41 personal cases, and attaches an extensive bibliography. He performs the operation of duodenojejunostomy in suitable cases. It is noteworthy that of his 41 cases no less than 22 had been operated on previously; of these, 8 had had gastro-enterostomy and 12 appendicectomy, presumably without relief of symptoms.

It would seem probable that in both the acute and chronic ileus a primary underlying obstructive cause is present at the site of the crossing of the mesenteric root, but that, in addition, in the acute cases a secondary toxic factor is added which overwhelms the patient and is responsible for the grave collapse seen in these cases, and also for the altered conditions of the stomach musculature and secretion. Such toxic symptoms are present, though in a greatly lessened degree, in the chronic cases, where they are manifested by headache, dizziness, malaise, and distaste for food. Alterations in the gastric and duodenal mucosa, as shown by hæmatemesis and ulceration, and malnutrition from stasis, are the chief local effects seen in the chronic cases.

Of the 7 cases on which this paper is founded, in 5 the symptoms appeared to be due to obstruction at the crossing of the mesentery. In these the hypertrophied and dilated stomach, widely patent pylorus, and dilated duodenum, ending abruptly at the site of the crossing of the mesenteric vessels, presented a striking picture at operation. In one of these cases there was a large chronic ulcer on the lesser curve. In the other 2 cases the obstruction was produced by the contraction of tissues lying in proximity to the duodenal wall. The symptoms in these cases were so similar to those presumed to be due to mesenteric constriction that they serve to strengthen the contention that the latter is a true cause of mechanical obstruction, and the primary condition accounting for the symptoms. In one of these cases (*Case* 6), the enormous dilatation of the stomach on admission, and the grave collapse and copious vomiting, closely resembled the condition seen in acute gastroduodenal ileus.

Symptoms.—The symptoms of digestive disturbance produced by chronic duodenal stasis usually show a gradual ingravescence over many years, with a tendency to acute exacerbations induced by dietetic indiscretions, posture, or even chronic constipation. During an attack epigastric pain is present, vomiting is common and often copious, and flatulence and epigastric distention are very marked.

Absorption from the toxic contents of the duodenum may cause headache or dizziness, or give rise to an icteric tinge of skin and conjunctivæ. Loss of weight may be considerable in the later stages of the condition. In some cases blood has been noticed in the vomit.

Patients with this trouble have often been submitted to surgical operation before the condition is recognized, the trouble being variously diagnosed in the appendix, the stomach, the duodenum, or the gall-bladder, as the symptoms may mimic disease of each of these organs very closely. Not only so, but the ileus may accompany chronic gastric and duodenal ulcer and gall-stones as a causative factor. The relationship of the onset of pain to the taking of food is not usually as definite as it is in cases of uncomplicated chronic gastric or duodenal ulcer. Nor is the pain so severe, being rather of the nature of intense flatulent discomfort. Vomiting is apt to be more copious and more frequent than is usually to be found with these conditions, except when they are associated with a high degree of pyloric stenosis; furthermore, vomiting does not give that relief to pain that it does in chronic gastric ulcer. The regurgitation of bile through the open pylorus that is

found in duodenal ileus is a very important diagnostic point, as it is nearly always absent in the vomiting of pyloric stenosis. During the attacks there are often nausea and distaste for food, though appetite may be normal at other times.

To sum up, the clinical history simulates to some extent that found in chronic inflammation of the appendix and gall-bladder, or chronic gastric or duodenal ulcer. The pre-operative diagnosis in the 7 cases recorded in this paper was accurate in 3 instances; in the 4 other cases the diagnosis was pyloric stenosis 2, gall-stones and chronic gastric ulcer 1 each. One of the cases had been operated on for chronic appendicitis before coming under my care.

X-ray Diagnosis.—Screening the patient after a barium meal may be of great assistance in making a diagnosis, as duodenal stasis and distortion may be definitely seen in some cases. In 2 cases out of 7, accurate diagnosis was made on this observation. At other times, however, the *x*-ray picture is not so clear, and in *Case* 1 the whole of the meal was held up in the stomach at the end of four hours, and the diagnosis of pyloric stenosis was consequently made. One other case had the large stomach with deep waves and a high degree of stasis suggestive of pyloric stenosis, but in this patient the condition was diagnosed on the great epigastric distention and copious vomiting of bilious material. In two of the cases there was some delay in emptying the stomach and some degree of pyloro-duodenal deformity. One case was reported as normal. Repeated observation of cases in which the symptoms are intermittent may be necessary to establish a diagnosis, and it is especially necessary to observe the duodenal loop immediately the meal enters the stomach and before the picture is obscured by jejunal filling, or the presence of an enlarged stomach in front of the loop.

Chemical Investigations.—In the course of investigating a number of cases presenting gastric symptoms by means of the fractional test meal, Dr. T. L. Hardy has made observations on three of my cases of duodenal ileus. In one, the quantity of fasting juice, the acid response, and the rate of emptying were normal. In a second case there was hyperacidity in the early stages, with efficient neutralization later, and a final rise to high values when the stomach was nearly empty. In a third case (*Fig.* 406) it was possible to make the diagnosis of duodenal obstruction with some confidence from chemical examination alone. Over a pint of dark-green turbid fluid, in which no starch was present, was extracted from the fasting stomach. All subsequent specimens contained much bile, while starch was present at three hours in considerable quantity. The acid values showed nothing noteworthy.

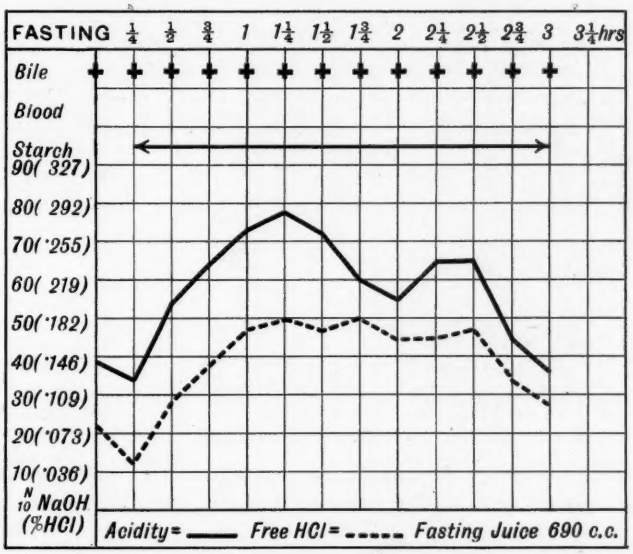

FIG. 406.—Fractional test meal in a case of chronic duodenal stasis (*Case* 7) due to cicatricial contraction involving the wall 4 in. from the pylorus.

The value of gastric analysis in these cases will clearly depend on the state of the pylorus. In the third case the duodenal obstruction was due to periduodenal contraction, the pylorus was patulous, and the stomach shared in the dilatation. The method is likely to be of value only in the more extreme types of the condition.

ILLUSTRATIVE CASES.

The following five cases present some of the salient features of the condition. In all, the mesenteric vessels appeared to be the main cause of the obstruction.

Case 1.—A man, age 35, who was serving in the Navy at the time of the onset of his illness and was otherwise healthy. His troubles commenced with severe colicky pain in the upper abdomen in 1916. The pain came on in attacks, was indefinitely related to food, starting from one to six or eight hours after a meal, and was accompanied by vomiting. He sometimes went as long as two months between attacks. In 1918 appendicectomy was performed; but though relieved for a while, in 1919 his attacks of pain and vomiting were again very severe. He vomited very large quantities, up to half a gallon. In November, 1919, he was again operated on at Chatham, adhesions being broken down and severe gastroptosis observed. In 1920 the old attacks recurred, and pain and vomiting were again severe. He was seen by me in 1921 for continuation of his pain and vomiting.

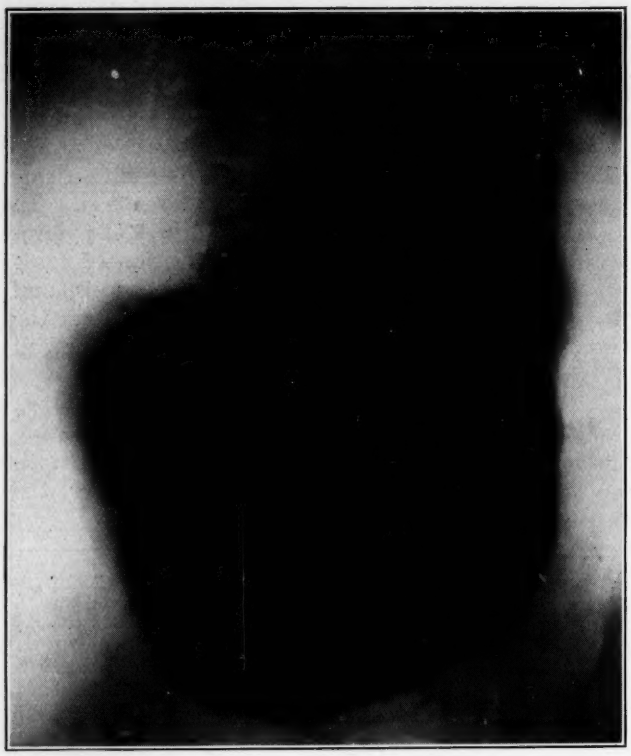

FIG. 407.—Radiograph of *Case* 1 taken four hours after barium meal. None of the meal had passed the pylorus, though it was widely open.

X-ray examination (*Fig.* 407) showed "typical pyloric stenosis, enlarged deep waves, all meal in stomach at end of four hours".

In view of his condition and these findings, laparotomy was decided on, and to my surprise the pylorus was much broader than usual and admitted three fingers easily; following down the duodenum it, too, was much wider than normal in its first, second, and third parts, and the dilatation terminated at the crossing of the superior mesenteric vessels, which stretched like a tight band across the bowel in its third part. Gastro-enterostomy was performed, as the operation of duodeno-jejunostomy was unknown to me at that time, and relief of the obstruction was urgently necessary, and gastro-enterostomy would seem to afford it. The result was successful, for with the exception of two large vomits during convalescence, the condition has been entirely relieved, and the patient has put on four stone in weight in fourteen months.

Case 2.—A woman, age 36. Symptoms of pain immediately after food, and frequent vomiting, for four years. Between 1919 and 1922 she had lost nearly three stone in weight, and was thin, pale, and ill-nourished on admission. She had localized tenderness to the right of the umbilicus, and in this area small peristaltic waves could be seen moving from left to right. The radiographic report stated that the stomach was small and quiet and there was slight pyloric deformity. In four hours one-tenth of the meal was still in the stomach. At operation the stomach was normal, except the pylorus, which admitted three fingers. The whole duodenum appeared dilated, for no apparent reason. On closer investigation, however, some adhesions were broken down which had cemented the dilated third part of the duodenum to the jejunum, and across the narrow isthmus of the bowel beneath these adhesions the superior mesenteric vessels were tightly stretched. Duodenojejunostomy was performed, and the appendix removed, as it was chronically inflamed. The patient made a satisfactory recovery.

It is interesting that in this case a large retroperitoneal pouch of peritoneum passed from the left side of the duodenojejunal junction for quite 2½ in. upwards and to the right behind the duodenum; this, if distended, must have increased the obstruction caused by the vessels. It is possible that the attacks were induced by herniation of bowel into this pouch.

This patient was operated on a year ago; she has now lost all symptoms of indigestion except some flatulence, and has recently given birth to a child. She has put on over a stone in weight.

Case 3.—A woman, age 50. For three years she has had attacks of epigastric pain and vomiting, the attacks getting more frequent and severe. During the attacks she has epigastric and left hypochondriac pain of a colicky nature, with frequent vomiting and great nausea and distaste for food. The vomiting does not relieve the pain, and the vomit contains bile. There is much flatulence. Between the attacks the appetite is good. There has been some loss of weight. Skiagram showed nothing abnormal in the stomach or duodenum. A pre-operative diagnosis of gall-stones was made.

At operation, the stomach and duodenum as far as the crossing of the mesenteric vessels were dilated and hypertrophied, and well-marked pressure on the third part of the duodenum by the mesenteric vessels was observed when the finger was passed beneath them. Technical difficulty prevented the performance of a duodenojejunostomy, as much bleeding ensued on attempting to mobilize the third part of the duodenum for its performance. A gastro-enterostomy was therefore performed. This patient, though better, still has nausea, and I feel it would have been better if a duodenojejunostomy could have been performed and the dilated duodenum drained at its distal extremity.

Case 4.—Male, age 33, who presented evidence of old rickets in stunted growth, kyphosis, and a pigeon breast. This patient had symptoms of chronic indigestion of twenty years' standing, with pain, vomiting, and occasional hæmatemesis occurring in attacks. He was admitted during one of these, and was thought to be bleeding from a chronic ulcer of the stomach or duodenum, as he had suffered recently from dizziness and fainting, and had melæna on admission. Vomiting had been more frequent recently, and did not relieve his pain: the vomit contained bile. *X*-ray examination failed to indicate the presence of ulcer, but some degree of stasis was present in the stomach. The appetite and nutrition were both poor, and he suffered from much flatulence.

Operation showed a hypertrophied and dilated stomach; the pylorus was 2 inches broad, and the duodenum dilated to the crossing of the mesentery, the bowel being normal beyond. General visceroptosis was present. Duodenojejunostomy was performed. At the end of three months he is much improved, but not wholly comfortable after meals. Weight is increasing.

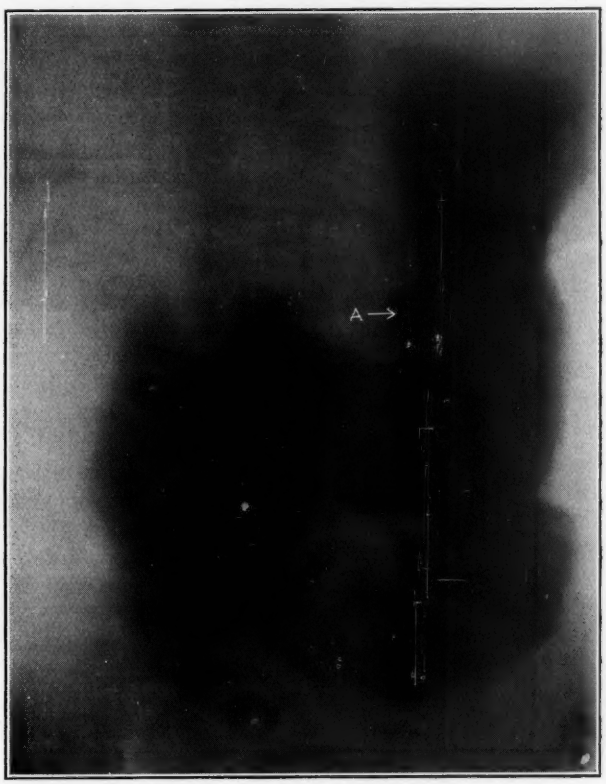

FIG. 408.—Radiograph of chronic gastric ulcer (A) in a case of chronic duodenal stasis due to pressure of the mesenteric vessels. Taken ten minutes after swallowing the meal. Obstruction is incomplete, as some barium has passed the duodenojejunal junction. The whole duodenum was dilated, but this was more evident with the screen at the moment of entry of the barium than is apparent in the picture. (*Case* 5)

Case 5.—**Chronic duodenal stasis with chronic gastric ulcer.**

Male, age 32, with a history of two months' severe indigestion. Up to the time of onset, patient states he was free from any digestive troubles. The onset was abrupt, with severe pain in the left hypochondrium, the pain coming on two hours after food, so that he was afraid to eat, though his appetite was good. Vomiting was frequent, especially at the onset of his illness; it relieved his pain, and he noticed that the vomit contained bile. His general nutrition was poor, and his mouth contained many septic stumps. Screen examination revealed a large chronic ulcer on the lesser curve (*Fig.* 408) and a considerable degree of duodenal dilatation throughout the length of the viscus.

At operation a chronic ulcer was found on the lesser curve 3 in. from the pylorus; its crater

easily accommodated the tip of the index finger when the stomach wall was invaginated into it; the pylorus was broad; the first part of the duodenum was 3 in. across, and the dilatation was so great in the third part that this retroperitoneal part of the gut was easily brought out of the abdominal cavity on turning up the colon. The dilatation ceased at the crossing of the mesenteric vessels. The jejunum was anastomosed to the third part of the duodenum, and the ulcer was left alone in the belief that it would heal when the stasis was relieved. This patient is doing well, but it is too early yet to say if the ulcer has been permanently cured by the relief thus afforded.

The two following cases present in their symptoms a picture very like that observed in those recorded above, and may be considered with them. In both of them obstruction of the duodenum was produced by contraction of the periduodenal tissues: in the one case by malignant disease, and in the other apparently by cicatrization secondary to some chronic adenitis in the neighbourhood of the viscus. The obstruction thus produced caused changes in the stomach and duodenum analogous to those seen in the cases due to mesenteric drag, though the development of symptoms was more rapid and lacked the characteristic intermissions of the latter cases. The close similarity between the symptoms seen in the two types of case affords, I think, strong evidence of the reality of the obstruction produced by tension of the mesenteric vessels where they cross the duodenum.

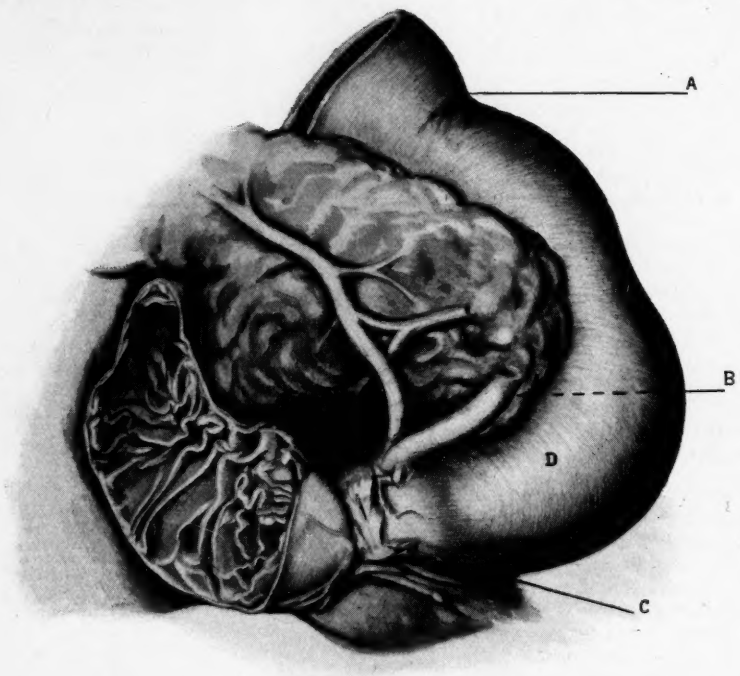

FIG. 409.—Duodenal obstruction by growth arising in the pancreas and invading the wall of the duodenum distal to entrance of common bile-duct. Seen from behind. (*Case* 6.) A, Pylorus; B, Common bile-duct; C, Growth; D, Distended loop of duodenum.

Case 6.—A domestic servant, age 32, who had presented no symptoms of digestive disorder up to the time of her present illness, was sent urgently to hospital in an extremely collapsed condition. Epigastric pain and vomiting had commenced suddenly two months previously, and vomiting had been frequent and copious ever since, especially so just previous to admission, so that when first seen the patient was almost moribund from loss of fluid. The abdomen was distended as by a large low tension cyst, and this, and the presence of a succussion splash, led to the passage of the stomach tube, which drew off 6 pints of brownish fluid. The house surgeon who saw her on admission stated that the tube could be felt in the stomach through the thin abdominal wall as low down as the brim of the pelvis. With daily lavage the patient improved and the vomiting ceased. Somewhat later, however, the vomiting again set in, and it was evident that if anything were to be done by operation it must not be delayed.

The radiographic report was as follows: "Stomach normal in size and shape; stasis in second part of duodenum; four hours, a fifth of contents was still in the stomach." The patient presented the appearance seen with acute gastroduodenal ileus—a feeble pulse, extreme lethargy, and cold

extremities—and although large quantities of fluid intravenously and subcutaneously improved her slightly, she was very feeble at operation. Laparotomy showed a stomach of moderate size with thick walls; the pylorus was broad and the duodenum very dilated. The dilatation ended at the middle line at a point which appeared near the termination of the duodenum; here a small hard mass of infiltrated tissue was present, and the front surface of the bowel was scarred over and contracted, the whole being not unlike a cotton-reel in size and consistence. The exact nature of the obstruction could not be determined, and so it was short-circuited by the rapid performance of a duodenojejunostomy.

The patient appeared little the worse for the operation, but gradually faded out in the next thirty-six hours. Post-mortem examination showed a thick-walled stomach of moderate size, and great dilatation of the duodenum as far as the obstruction, where the bowel was embraced by a mass of cicatricial tissue (*Fig.* 409). Closer examination of the site of the obstruction showed that it was at or near the entrance of the common bile-duct, which was apparently lower down in the duodenum than normal. The tissue causing obstruction was mainly fibrous, but areas of a spheroidal-celled carcinoma in the fibrous mass indicated that a scirrhous growth of the pancreas was probably the primary cause of the obstruction.

Case 7.—**Periduodenal fibrosis around the second part of the duodenum: obstruction.**

A male, age 52, had sudden onset of pain and vomiting, which continued with increasing severity up to the time of admission fourteen days later. The pain was severe and unrelated to food, and was epigastric in site. Vomiting was frequent and copious, and took place at indefinite times of the day and night, and the vomit contained bile. His general nutrition was good, though he stated he had lost three stone in weight in the last two years. He had much epigastric distention, and the outline of a hypertrophied and dilated stomach could apparently be felt coming low down in the epigastrium. *X*-ray examination showed an enlarged stomach with deep waves, and at the end of four hours a third of the meal was in the stomach. A diagnosis of duodenal obstruction was made on clinical signs.

At operation, the stomach, and the first and second parts of the duodenum, were found to be greatly hypertrophied and dilated; the pylorus easily accommodated three fingers; there was no dilatation of the third part. The dilatation ended 4 in. from the pylorus in a cicatricial ring, from which a small portion of tissue was removed for histological examination. The pathological report on this showed it to be dense fibrous tissue only, and there was no evidence of its origin.

As the obstruction was so high up it was decided to do a gastro-enterostomy; to bring a loop of jejunum so far across to the right, in order to do a duodenojejunostomy, would, it was feared, result in causing embarrassment to the function of the loop. Convalescence was marred by several bouts of vomiting in the first week, but otherwise the patient made a good recovery.

Cases 6 and 7 belong to a well-recognized but comparatively small group in which duodenal obstruction is caused by involvement of the second or third parts of the bowel in neighbouring inflammations or growths, and have only been mentioned because of the similarity of the picture they present to the cases of obstruction by the crossing of the mesenteric root. Of obstruction caused by the latter method the five first cases were well-marked examples; but it seems probable from observations in the course of performing laparotomy for other conditions that duodenal dilatation of lesser degree is not at all uncommon; and though in these cases the condition is not sufficiently marked to call for surgical relief, yet it may be a factor in producing disturbance of function in the stomach, duodenum, or biliary tract.

The cases quoted were so well established that there can be no doubt they were wholly responsible for the patients' condition, and merit recognition as a clinical entity, definitely to be borne in mind when investigating a case of chronic indigestion. Similarly, when performing laparotomy for this condition, a routine examination of the third part of the duodenum must be made after the examination of stomach, gall-bladder, appendix, and other possible sources of trouble.

Treatment.—The relief that is afforded in acute gastric dilatation by lavage and posture suggests the possibility of using these measures in the chronic condition. Furthermore, it is possible that abdominal support by a suitable belt may be of value in relieving the visceroptosis so commonly found with the condition. In cases, however, in which the obstruction has reached a certain degree of severity, permanent relief can probably only be obtained by operation. In these cases the stasis is such that from time to time the hypertrophy of the stomach and duodenum fails to overcome the block, compensation fails, and hypertrophy gives way to dilatation. When this stage is reached, relief is permanently given by short-circuiting the duodenum in its third part into the jejunum,

close to its commencement. This procedure was suggested by Barker[14] in 1906, but the first case recorded in which it was carried out was by Stavely[15] in 1908 ; full details of the operative procedure are given in Kellogg's paper.[13] The operation resembles technically a gastro-enterostomy, but is a little more difficult to perform, as the duodenum is retro-peritoneal and cannot be brought to the surface as easily as the stomach. The dilatation of the horizontal part of the duodenum which is so noticeable in the established cases renders the operation easier than might be expected. In *Case* 5, for instance, this part of the duodenum could easily be brought out of the abdomen, and the operation be performed with as much ease as the ordinary gastro-enterostomy. In three cases a gastro-enterostomy was performed : in the first, because the operation of duodenojejunostomy was unknown to me ; in *Case* 3 an attempt to perform the operation of duodenojejunostomy was frustrated by considerable hæmorrhage from large veins in front of the dilated duodenum ; whilst in *Case* 7 the obstruction was so high up in the duodenum that it appeared preferable to do a gastro-enterostomy.

Convalescence in these three cases was unsatisfactory owing to vomiting, which was of the nature of that seen in the vicious circle after gastro-enterostomy, and was relieved by lavage. *Case* 1 has done so well since leaving hospital that it would appear gastro-enterostomy may occasionally give permanent relief to the condition if the initial post-operative dangers of the vicious circle are tided over.

Of the other two cases in which gastro-enterostomy was performed, one is definitely unsatisfactory ; the other is relieved by the operation, but is too recent to enable one to judge of the permanency of the relief. There is no doubt that duodenojejunostomy, by relieving the dilated bowel at its distal end, is the operation of election, whilst gastro-enterostomy is liable to be associated with a post-operative vicious circle.

CONCLUSIONS.

1. The condition of duodenal ileus may arise from pressure of the mesenteric root containing the superior mesenteric artery and vein on the third part of the duodenum.

2. Whilst probably such obstruction is the chief predisposing cause of acute dilatation of the stomach, it may also give rise to chronic digestive disturbance, simulating chronic ulcer of the stomach and duodenum, or inflammation of the appendix or gall-bladder, and is sufficiently common to be recognized as a clinical entity.

3. Apart from the direct effects produced by obstruction, duodenal ileus plays a part in the etiology of some cases of chronic gastric and duodenal ulceration and infections of the biliary tract.

4. In many cases an accurate pre-operative diagnosis of the condition may be made by a careful consideration of the clinical symptoms and the results of a radioscopic examination.

5. When performing laparotomy the condition must be borne in mind and sought for as a cause of chronic indigestion, and, in the presence of hypertrophy and dilatation of the stomach and duodenum with patent pylorus, the operation of duodenojejunostomy performed.

REFERENCES.

[1] WILKIE, D. P. D., *Brit. Jour. Surg.*, 1921, No. 34, 204.
[2] FRAZER, J. E., *Jour. of Anat.*, 1919, liii, July, 292.
[3] ROKITANSKY, *Lehrbuch der path. Anat.*, 1863, iii, 3 Aufl.
[4] GLÉNARD, ' De l'Enteroptose ', *Presse méd. Belge*, 1889, xli, 57.
[5] ALBRECHT, P. A., *Virchow's Archiv. f. path. Anat.*, clvi, 285.
[6] ROBINSON, B., *Lancet-Clinic*, Cincinnati, 1900, xlv, 577.
[7] CONNER, L., *Trans. Assoc. Amer. Phys.*, 1906, xxi, 579.
[8] BLOODGOOD, J. C., *Ann. of Surg.*, 1907, xlvi, 736.
[9] CODMAN, E. A., *Boston Med. and Surg. Jour.*, 1908, clviii, 503.
[10] WIPPLE, STEIN, and BERNHEIM, *Jour. Exper. Med.*, 1913, xvii, 286, 323.
[11] SWEET, PEET, and HENDRIK, *Ann. of Surg.*, 1916, June, 720.
[12] ELLIS, J. W., *Ibid.*, 1922, lxxv, 429.
[13] KELLOGG and KELLOGG, *Ibid.*, 1921, lxxiii, 578.
[14] BARKER, *Johns Hop. Hosp. Bull.*, 1906, 37.
[15] STAVELY, *Ibid.*, 1908, 252.

ABNORMALITIES OF THE RIGHT HEPATIC, CYSTIC, AND GASTRO-DUODENAL ARTERIES, AND OF THE BILE-DUCTS.

By E. R. FLINT, Leeds.

Human beings are singularly alike in their general anatomical construction; yet when we come to investigate one particular region with more detail it is surprising how frequently we meet with variations of one sort or another. More especially does this apply to the vascular system, and in no region more than to the liver. This is, I think, generally appreciated by anatomists. There are, however, variations in the excretory ducts of the liver almost as frequently, and anatomists have not given the attention to this subject that a part, so important from the surgical point of view, deserves.

I have made 200 dissections on post-mortem subjects of the vessels and ducts of the liver. The dissections were made as far as possible consecutively. There has been no selection of cases, and it is claimed, therefore, that the anatomical details given are a very fair representation of the state of the parts as found in the human subject, and that the results of anyone undertaking so large a number of dissections will be found to corroborate mine. In addition, I have paid particular attention to these parts during operations, but I have not included the results in the statistics; I shall refer to them later.

If we exclude the appendix, the gall-bladder and its ducts more often call for operative treatment than any other intra-abdominal viscus, and a great deal of this surgery is very badly done, if one may judge by the great number of cases one sees which require a second operation, mostly for reasons that should not arise if proper care and knowledge had been applied at the first attempt. Technically, gall-bladder surgery is much the most difficult of any abdominal surgery, and inadequate appreciation of the abnormalities of this region does not lessen the risks.

The arrangement of the vessels and ducts given as normal in the text-books of anatomy is shown in *Fig.* 410. In my series of 200 cases, only 69 conform to this type. So frequent are variations that it is impossible to regard any one type as normal; the arrangement found in the 69 cases can only be described as the most usual one.

I will describe first the vessels as found in my series.

The Right Hepatic Artery.—This arises from the main hepatic trunk in 158 cases, and to reach the liver passes behind the common hepatic duct in 136 (*Fig.* 410), and in front of this duct in 25 (*Fig.* 411). In 42 the right hepatic artery arises from the superior mesenteric artery (*Fig.* 412), and always passes behind the common duct. In 7 cases there are two right hepatic arteries, one from the hepatic trunk and one from the superior mesenteric (*Fig.* 413). In 2 cases there are two right hepatics both from the main hepatic, one passing in front of, and the other behind, the common hepatic duct. In 4 cases, in addition to passing behind the ducts, the main hepatic or the right hepatic artery also passes behind the portal vein (*Figs.* 414 *and* 432).

I am aware that the right hepatic artery very occasionally arises from the aorta, the right renal, the gastric, or the inferior mesenteric artery, but there are no instances in my series.

The right hepatic artery, as it crosses the bile-duct in 25 cases—especially when crossing low down near the junction of the cystic duct—is liable to injury during cholecystectomy. There are two other variations which render it even more liable to an accident:—

1. In 8 of the cases, all in elderly people, the artery is tortuous and projects forwards to the right of the common hepatic duct, something like the hump of a caterpillar's back during progression (*Figs.* 413 *and* 425). From the summit of this hump may arise the

DIAGRAMS ILLUSTRATING VARIOUS ABNORMALITIES IN THE ARTERIES AND BILE-DUCTS MET WITH IN GALL-BLADDER SURGERY.

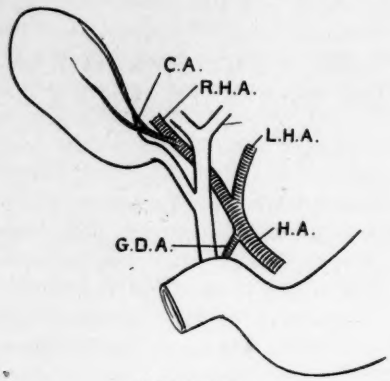

FIG. 410.

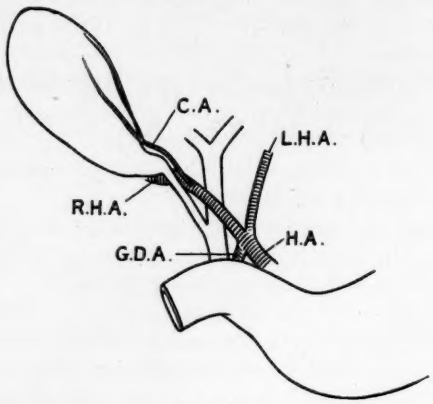

FIG. 411.

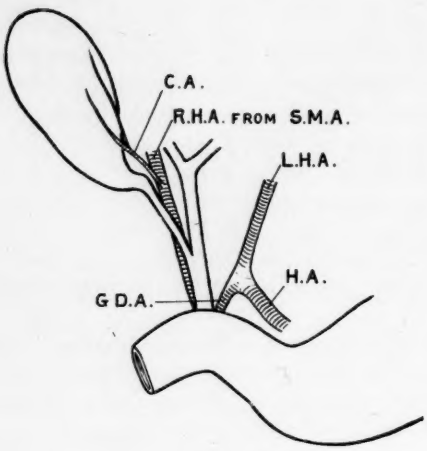

FIG. 412.

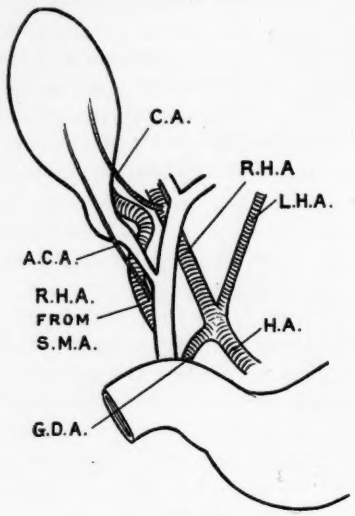

FIG. 413.

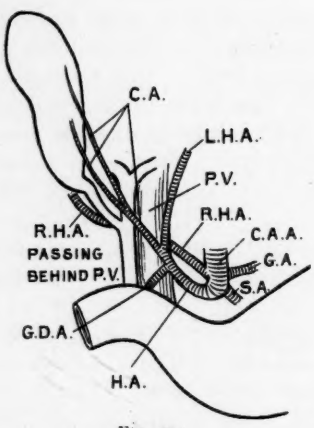

FIG. 414.

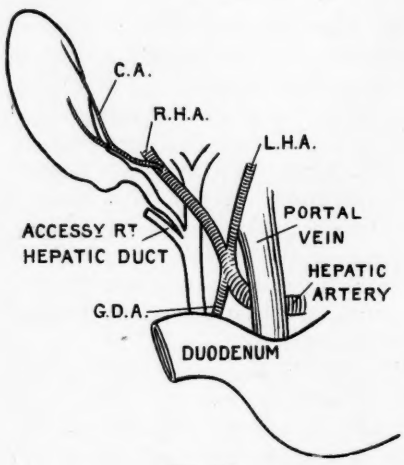

FIG. 415.

(C.A.) Cystic artery; (G.D.A.) Gastroduodenal artery (H.A.) Hepatic artery; (L.H.A.) Left hepatic artery; (R.H.A.) Right hepatic artery; (S.M.A.) Superior mesenteric artery: (A.C.A.) Accessory cystic artery; (P.V.) Portal vein; (C.A.A.) Cœliac axis artery; (G.A.) Gastric artery; (S.A.) Splenic artery; (R.H.D.) Right hepatic duct; (S.P.D.A.) Superior pancreatico-duodenal artery; (C.D.) Cystic duct.

cystic artery, and on pulling up the gall-bladder the projecting artery comes to lie at a level which is even a little anterior to that of the cystic duct. I have known this mistaken for an enlarged cystic gland; fortunately the error was recognized in time. I have no doubt, however, that it has been included in the clamp from time to time.

2. The right hepatic artery may run parallel, and very close, to the cystic duct and the neck of the gall-bladder, almost suggesting a double cystic duct (*Fig.* 424). It could very easily be included in the clamp applied to the duct.

The Cystic Artery arises from the right hepatic 196 times out of the 200; in 3 from the left hepatic (*Figs.* 418 *and* 434), and in 1 from the gastroduodenal artery (*Fig.* 417).

In 32 cases it passes in front of the common hepatic duct (*Figs.* 414 *and* 416), and in 168 it arises just to the right side of the common hepatic duct (*Fig.* 410), or behind it (*Fig.* 436). The former is much the more common. This difference in point of origin has some surgical interest to which I will refer later.

Accessory Cystic Artery.—In 31 cases there is an accessory cystic artery. There is, therefore, a single cystic artery in only 169. Attention is not called to this in anatomy books. The accessory cystic artery arises from the right hepatic in 16 (*Figs.* 413, 419, *and* 420), from the left hepatic in 3 (*Figs.* 421 *and* 434), from the gastroduodenal in 11 (*Fig.* 422), and from the superior pancreaticoduodenal in 1 case out of the 200 (*Fig.* 423). In the last two instances it is liable to injury during the operation of cholecdochotomy. When there was an accessory cystic artery I found that it invariably crossed in front of the bile-ducts. Ignorance of the occurrence of accessory cystic arteries may be responsible for rather severe hæmorrhage.

There are three other points of interest about the arteries.

1. In opening the common duct it is quite common to have an annoying hæmorrhage from an artery which crosses the front of the supraduodenal part of this duct. Though a plexus of veins and arterioles is described on the surface of the duct, I have seen no mention of this artery in the literature. Surgeons know it well, and I was curious, therefore, to discover its source. I am unable to give the frequency of its existence, as I did not begin to look for it from the first, but I found it quite often; it arises from the hepatic artery low down, or from the superior pancreaticoduodenal, or from the gastroduodenal, and runs a rather tortuous course along the anterior surface of the duct (*Figs.* 425 *and* 428). It may be the superior pancreaticoduodenal itself, when this vessel comes off higher than usual.

2. The gastroduodenal artery in a small percentage of cases forms a curve in front of the lower supraduodenal part of the common duct (*Fig.* 426), and might be wounded in opening the common duct. Also the superior pancreaticoduodenal artery occasionally crosses the duct just above the level of the upper border of the duodenum (*Figs.* 423 *and* 425).

3. Although this paper is chiefly concerned with the right hepatic artery, I should like to mention one point about the left hepatic. In my first 100 cases I dissected out this artery, and found it was quite common to have two left hepatic arteries. In 32 cases one artery came from the main hepatic and the other from the gastric, and in one case the only artery which could be found going to the left lobe of the liver came from the gastric. In doing a gastrectomy this vessel would almost certainly be divided, and troublesome bleeding might occur from the distal end.

The Bile-ducts.—The second part of this paper deals with the bile-ducts. According to the anatomical text-books, the right and left hepatic ducts unite in the portal fissure, or just beyond it, to form the common hepatic duct. This structure is from 1 in. to 1¼ in. long. The cystic duct is from 1 in. to 1½ in., and uniting with the common hepatic duct at an acute angle, they together form the common bile-duct, which is about 3 in. long. The length of the supraduodenal part of the common duct varies with the level of the duodenum and the point at which the cystic and common hepatic ducts join; the average length for this part of the duct is held to be about one-third of the whole length of the common duct.

DIAGRAMS—*continued.*

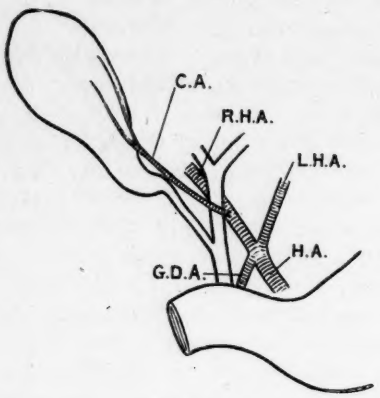

FIG. 416.

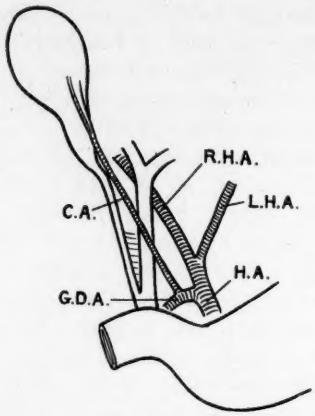

FIG. 417.

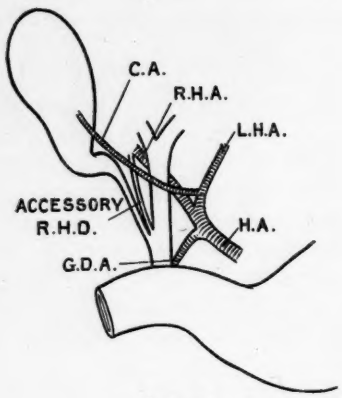

FIG. 418.

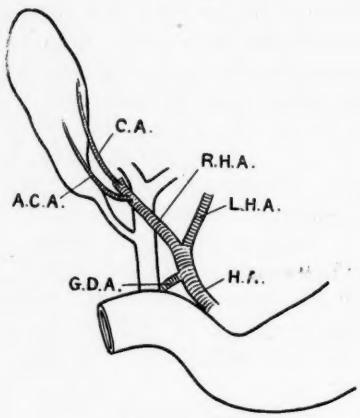

FIG. 419.

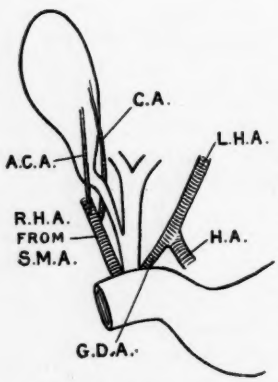

FIG. 420.

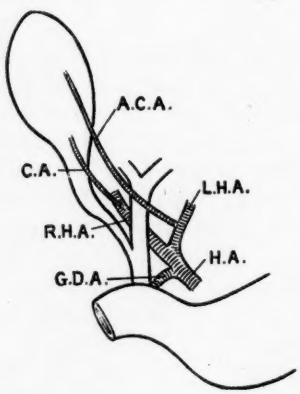

FIG. 421.

(C.A.) Cystic artery; (G.D.A.) Gastroduodenal artery; (H.A.) Hepatic artery; (L.H.A.) Left hepatic artery; (R.H.A.) Right hepatic artery; (S.M.A.) Superior mesenteric artery; (A.C.A.) Accessory cystic artery; (P.V.) Portal vein; (C.A.A.) Cœliac axis artery; (G.A.) Gastric artery; (S.A.) Splenic artery; (R.H.D.) Right hepatic duct; (S.P.D.A.) Superior pancreatico-duodenal artery; (C.D.) Cystic duct.

Now, though it is true that the cystic and common hepatic ducts do come together at such a point as to give an average of lengths as stated, they do not unite here. Almost always they are merely bound together by fibrous tissue, and by dissection it is possible to separate them from each other for a few millimetres to as much as 2 in. or more. The most common point, according to my observations, at which union actually occurs is within 1 cm. of the upper border of the duodenum (*Fig.* 417).

In 28 cases there was no supraduodenal common duct at all (*Fig.* 429), the union occurring at a point anywhere from behind the upper border of the duodenum to the part embedded in the pancreas, and in 3 cases the only representative of the common duct was that part which lies in the wall of the duodenum (*Fig.* 430). I did not obtain a specimen of separate entrance of the cystic and common hepatic ducts into the duodenum, though this does occasionally occur.

It is usual for the cystic duct to open on the right side of the main bile-duct; but in a certain proportion of cases it enters on the front, the back, or even the left side, taking a spiral course around the main duct. In 8 of my cases the junction was on the front aspect of the duct (*Fig.* 431), and in 3 on the posterior. In one of the latter the union was so far around the back of the duct as to be almost on the left side (*Fig.* 432).

Accessory Bile-ducts.—The most interesting abnormality of the ducts is the presence of an accessory one. I have 29 examples of this. All of them are accessory right hepatic ducts. The duct leaves the liver at the extreme right end of the portal fissure, and, lying at first on a rather deeper plane than the cystic duct, joins the extrahepatic ducts anywhere between the junction of the right and left hepatic ducts and the point at which the cystic duct opens into the main duct. It usually has the same relation to the right hepatic artery as the normal ducts—that is, the artery passes behind the duct.

There is no reference in the anatomy books to an accessory duct such as I found. It is true they speak of a junction of the right and left hepatic ducts at varying levels, but in all my cases this junction was at or near the normal level, and the ducts I am describing were additional. Eisendrath[1] gives drawings of these accessory ducts, but I gather from the text that they were not taken from his dissections. I have seen no reference to these ducts in the surgical literature, with the possible exception of a case of Kehr's in which he speaks of wounding the right hepatic duct where it was making a low junction with the left hepatic. It is possible this was an accessory duct as described by me, mistaken by him for the right hepatic duct.

I have classified these accessory ducts into three types, according to the level at which they enter the main duct. This is done from a surgical rather than an anatomical standpoint.

1. The junction occurs in the upper half of the common hepatic duct or in the right hepatic duct (*Figs.* 433 *and* 434). There are 9 of these. In this type the union is so high up that the duct is unlikely to be of surgical importance.

2. The junction occurs in the lower half of the common hepatic duct (*Fig.* 435). There are 9 cases in this class also. The union is so near that of the cystic and common hepatic ducts as to be definitely in the field of a cholecystectomy operation.

3. The junction is at the union of cystic and common hepatic ducts (*Figs.* 415, 436, 437). There are 10 of these cases. The junction is usually in the actual angle of the cystic and common ducts, but may be in the extreme lower end of the cystic duct, or in the extreme lower end of the common hepatic duct. In any case it is difficult to see how the duct could be avoided whilst clamping the cystic duct unless its presence had been previously detected.

In one specimen (*Fig.* 438) the accessory duct leaves the right hepatic duct and enters the cystic duct, and of course must be cut during cholecystectomy.

The size of these accessory ducts varies. The smallest is only large enough to admit a good-sized bristle. The largest is as big as the right hepatic duct. The commonest size is about half-way between these limits; that is to say, it is quite a considerable duct.

There is one other abnormality of the ducts in this series, which is a curiosity rather than one of practical interest, and that is a congenital obliteration of the ducts. No

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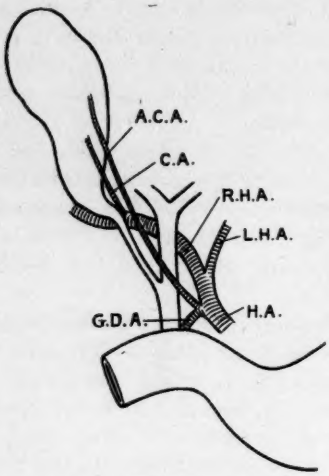

FIG. 422

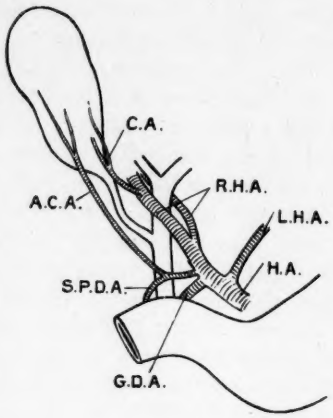

FIG. 423.

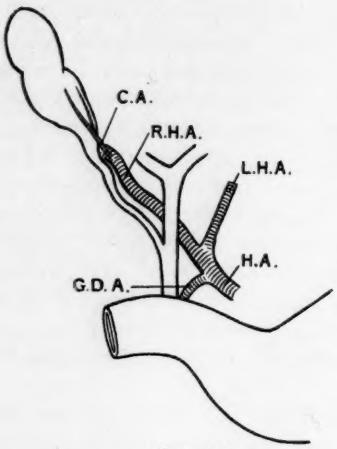

FIG. 424.

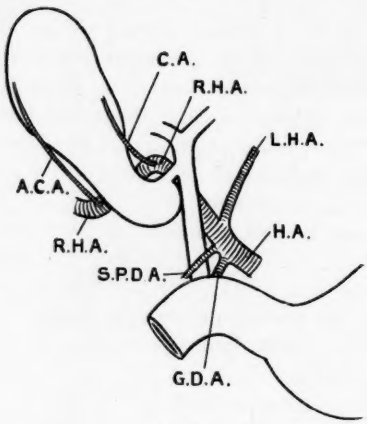

FIG. 425.

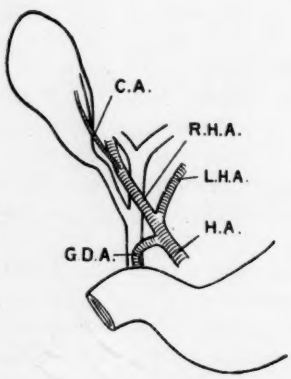

FIG. 426.

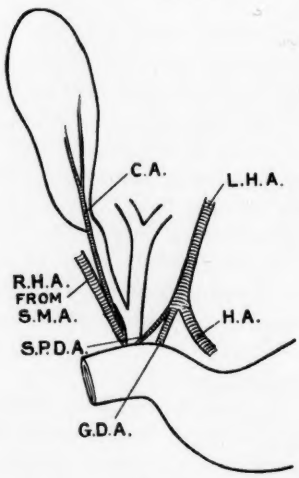

FIG. 427.

(C.A.) Cystic artery; **(G.D.A.)** Gastroduodenal artery; **(H.A.)** Hepatic artery; **(L.H.A.)** Left hepatic artery; **(R.H.A.)** Right hepatic artery; **(S.M.A.)** Superior mesenteric artery; **(A.C.A.)** Accessory cystic artery; **(P.A.)** Portal vein; **(C.A.A.)** Cœliac axis artery; **(G.A.)** Gastric artery; **(S.A.)** Splenic artery; **(R.H.D.)** Right hepatic duct; **(S.P.D.A.)** Superior pancreatico-duodenal artery; **(C.D.)** Cystic duct.

bile-ducts or definite gall-bladder could be found. The infant lived thirteen weeks, having had jaundice from birth, and having passed only clay-coloured stools. At the post-mortem examination the liver was found to be fibrosed (*Fig.* 439).

THE SURGICAL SIGNIFICANCE OF THESE ABNORMALITIES.

Practically all the accidents to the ducts and vessels occur during the operation of cholecystectomy with or without choledochotomy, and since cholecystectomy has almost entirely displaced cholecystostomy it is obviously the duty of every surgeon to make himself familiar with both the normal and abnormal anatomy of these parts.

It is customary, for several reasons, to begin the removal of the gall-bladder at the cystic duct, and it is here that all the traps lie. There is only one way to avoid catastrophes, that is to fix the neck of the gall-bladder with a clamp, and, after snicking through the gastrohepatic omentum near this point, gently to wipe the fatty tissue towards the common duct. The cystic duct and artery now come clearly into view and can be separately secured. If there should be an accessory artery or duct it will be exposed to view before being divided, and the junction of the cystic duct with the main duct can be seen distinctly. There are a few cases, however, in which everything is so deformed, thickened, and contracted by inflammation, that it is not possible to obtain a really clear definition; it is in this class of case that the most expert surgeons have probably all had unhappy experiences.

Secondary operations on gall-bladder cases have to be performed much too often, for I venture to say that 99 out of 100 could be avoided by a proper definition of the parts at the original operation, combined with a better knowledge of the pathology of the gall-bladder. The commonest blunders committed at the primary operation are: (1) Leaving the gall-bladder, through an inadequate appreciation of the earlier signs of cholecystitis; (2) Overlooking a stone in the cystic or common duct; (3) Injury to the common, or common hepatic, duct; (4) Injury to an accessory hepatic duct.

1. The first group is beyond the scope of this paper.

2. In the second group, difficulty might arise through the spiral arrangement of the cystic duct referred to above. A stone in that part of the duct which lies behind or in front of the common duct might be missed, or to expose it the common duct might be opened unnecessarily. Anyone unacquainted with this anomaly would be much perplexed on finding that he had opened two ducts to get at a stone which he had expected to find in the common duct. A stone in the normally disposed cystic duct or in the common duct should never be overlooked after a proper exposure of the parts.

I have already alluded to the abnormal arteries which may be encountered in opening the common duct. The superior pancreaticoduodenal and gastroduodenal arteries may be wounded in the transduodenal method of opening the common duct, for either vessel may lie in front of the duct just above the ampulla of Vater.

3. The injuries inflicted on the common hepatic or common duct practically always occur for one reason, that of not seeing clearly the various structures before applying a clamp. It is astonishing the number of surgeons who habitually neglect this simple precaution. Should the structures not be clearly defined, there are four causes to which injury to these ducts may be due: (1) A large sigmoid curve of the gall-bladder may lie closely attached to the upper part of the common duct, and on pulling up the gall-bladder the duct comes with it and may be taken for the cystic duct. In this case the common duct will be cut right across. (2) On pulling up the gall-bladder where there is a very short cystic duct, a V-shaped segment of the main ducts will be drawn up as well, when possibly as much as 1 in. of the common hepatic and common ducts may be removed. (3) A clamp may be applied rather beyond the cystic duct so that a bite is taken from the side of the main duct. (4) The forceps on the cystic artery may pull off, and hasty efforts to secure it again may mean injury to the common hepatic duct through grasping the whole or a part of it with the vessel. More especially is this likely to occur when the artery arises from the right hepatic behind the duct, as it does in a small number of cases, for under these circumstances the vessel retracts out of sight.

DIAGRAMS—*continued.*

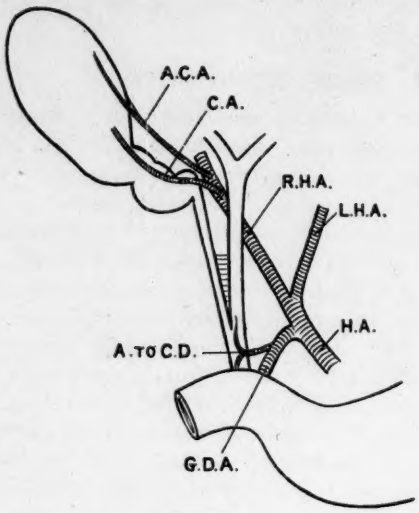

FIG. 428.

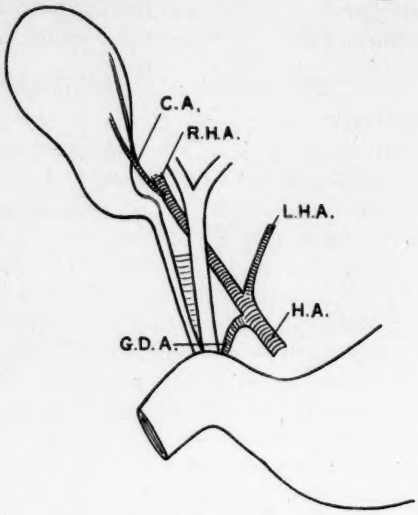

FIG. 429.

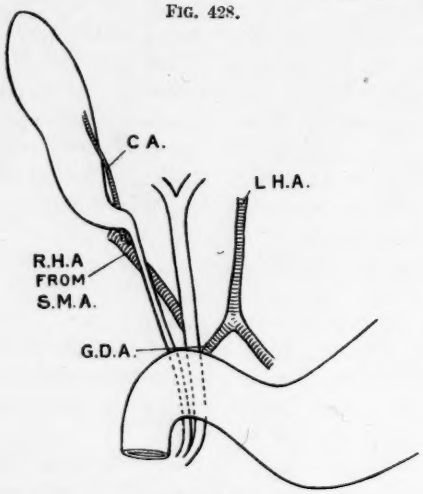

FIG. 430.

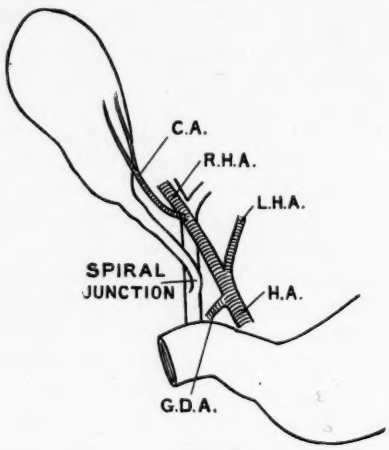

FIG. 431.

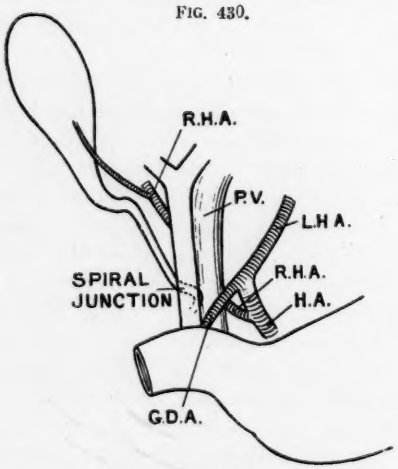

FIG. 432.

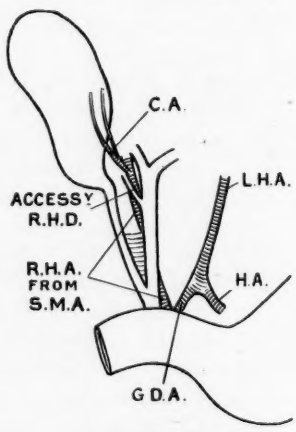

FIG. 433.

(C.A.) Cystic artery; (G.D.A.) Gastroduodenal artery; (H.A.) Hepatic artery; (L.H.A.) Left hepatic artery; (R.H.A.) Right hepatic artery; (S.M.A.) Superior mesenteric artery; (A.C.A.) Accessory cystic artery; (P.V.) Portal vein; (C.A.A.) Cœliac axis artery; (G.A.) Gastric artery; (S.A.) Splenic artery; (R.H.D.) Right hepatic duct; (S.P.D.A.) Superior pancreatico-duodenal artery; (C.D.) Cystic duct.

All these injuries may be successfully repaired at the time, if recognized. But they may not be revealed, for the very reason which was responsible for the catastrophe; or, being repaired, a stricture may follow. Another operation will then be required if the patient survive, and anyone who has undertaken this type of operation knows how exceeding difficult it may be.

4. In the fourth group the accessory ducts are injured. There is no literature on this subject that I know of, for I do not think surgeons as a whole are aware that these ducts exist, and certainly they are not in the habit of looking for them at operation. I have already mentioned the one possible exception of Kehr's.

I believe, for reasons which I will give presently, that these ducts are injured as often as the common duct, if not more frequently.

Since I have interested myself in this subject I have seen an accessory duct three times at operation during identification of the structures in the region of the cystic duct preparatory to clamping it. Before this I had two or three times seen the open end of what appeared to be a duct without being able to determine what it was; the end was ligatured and the patients recovered normally. Sir Berkeley Moynihan tells me he has had a similar experience. In one of my cases I saw bile coming from the cut duct and was much perturbed, as I felt sure I had divided the common hepatic duct, but investigation showed this structure to be intact.

In order to strengthen my contention of the importance of these ducts to the surgeon, I have been through the post-mortem records of the cases dying after cholecystectomy at the Leeds General Infirmary during the years 1908 to 1922 inclusive. During this period there were eight deaths due to bile leaking into the peritoneal cavity in considerable quantities. They were all cases of cholecystectomy without an accompanying choledochotomy. One of these deaths, which occurred in 1917, is very instructive, giving the evidence for which I was looking, and proving conclusively the importance of determining whether or no an accessory bile-duct is present. This is the post-mortem report: "On opening the abdomen a considerable quantity (one pint) of bile is found in the flank, pelvis, and around the liver. The gall-bladder has been removed. The ligature on the cystic duct is intact. Close to the liver (but not in connection with the hepatic ducts proper) is seen an open bile-duct, and, on squeezing, bile exudes from it. There is nothing else of note."

In the other seven cases an extravasation of a considerable quantity of bile was noted, but the source was not determined. I think it is highly probable that some, if not all, of these cases had a divided accessory duct as in the case reported. It is more than probable that the source would be overlooked at post-mortem, for the presence of these ducts is not generally known, and therefore would not be specially sought for.

After cholecystectomy with drainage, a small percentage of patients discharge bile from the wound, though the cystic duct has been ligatured. This begins at once or within a few hours of the operation, and is obviously bile from a duct. It has been attributed to bile from the raw surface of the gall-bladder bed; but it is too profuse for that, and, moreover, is not intimately mixed with blood as it should be if from this source. The other explanation given is that the ligature has slipped off the cystic duct. This has never seemed to me an adequate explanation, for the cystic duct is easy to tie, and there is only a very low pressure behind the ligature. In view of what has been said above, a much more reasonable supposition is that of an injured accessory duct.

Though it is bad surgery to injure these ducts, the probability is that little harm would result in the majority of cases, provided a drainage tube were left in. Many surgeons have advocated from time to time closure of the abdomen after cholecystectomy. Probably the fashion is more prevalent now than ever before. I suspect these surgeons are not aware of the possibility of the presence of accessory ducts, as the subject has never been raised in this connection, and closure of the abdomen with one of these ducts cut would be a disaster. Such an accident would in all probability be explained as a leak from the cystic duct.

To this the argument might be advanced by those who favour closure that bile would

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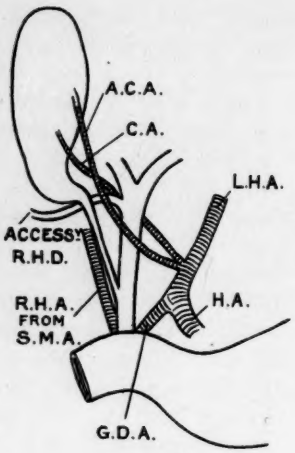


FIG. 434.

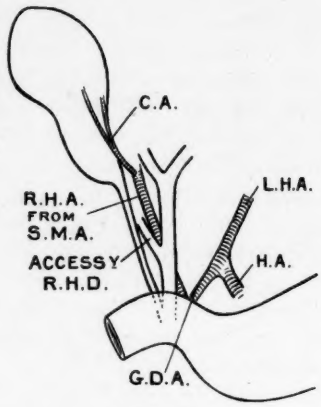


FIG. 435.

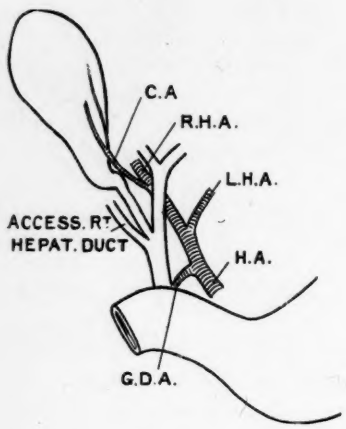


FIG. 436.

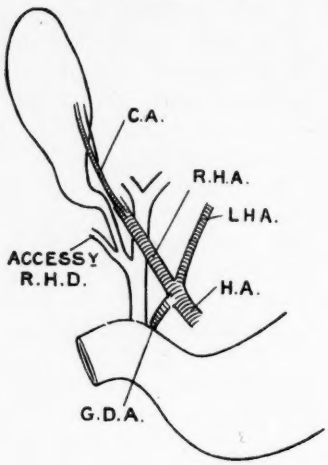


FIG. 437.

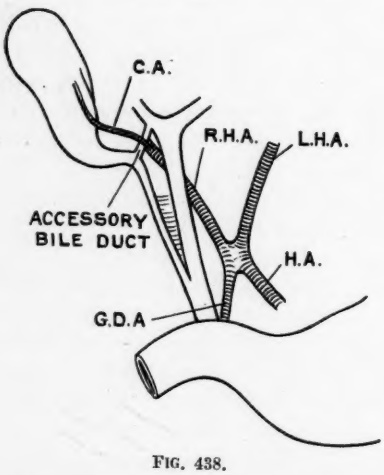


FIG. 438.

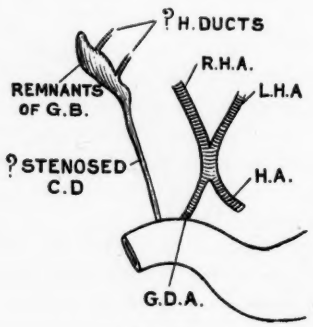


FIG. 439.

(C.A.) Cystic artery; (G.D.A.) Gastroduodenal artery; (H.A.) Hepatic artery; (L.H.A.) Left hepatic artery; (R.H.A.) Right hepatic artery; (S.M.A.) Superior mesenteric artery; (A.C.A.) Accessory cystic artery; (P.V.) Portal vein; (C.A.A.) Cœliac axis artery; (G.A.) Gastric artery; (S.A.) Splenic artery; (R.H.D.) Right hepatic duct; (S.P.D.A.) Superior pancreatico-duodenal artery; (C.D.) Cystic duct.

be seen to flow from the open end of the duct during the operation, and that the application of a ligature would make it safe to complete the abdominal closure. But this does not necessarily follow, for it is well known that after putting a tube in the common duct not a single drop of bile may appear sometimes for twenty-four or thirty-eight hours. Though this is not so likely to happen after cholecystectomy alone, I have no doubt that in those cases associated with much hepatitis the secretion of bile is more or less temporarily suspended. Moreover, there is no objection of any moment to leaving a piece of dental rubber as a drain for one or two days through an incision which displaces the rectus outwards. I have never seen a hernia result, and it will save a life now and again.

Two interesting questions arise to which no definite answer can be given as yet :—

1. What happens to that part of the liver drained by one of these ducts after a ligature has been applied ?

2. What becomes of the duct if it be divided and left open ?

As to the first question, only a limited part of the liver drains into this duct, and one would expect the healthy organ would be able to compensate in its other parts, for the capacity of the liver-cells to multiply in accordance with necessity is equalled by no other organ in the body. I performed an experiment with the object of finding out whether bile cut off in one part of the liver could make its way round to another. I ligatured the common duct low down, and also the cystic and left hepatic ducts. I then injected methylene blue into the upper part of the common duct ; the only way into the liver was up the right hepatic duct, and yet bile appeared in both the right and left lobes. I have not had time to follow up this investigation, and, as I did not know the exact pressure at which the dye was injected, too much importance must not be attached to it. The subject is, however, worth further investigation, for it is known that bile can make its way from one group of liver-cells to another in the immediate neighbourhood.

Suppose that one of these accessory ducts were ligatured when the liver was not healthy, as, for instance, in a case of prolonged obstruction to the common duct by a stone. In such a case temporary suppression of bile is known to be not uncommon after operation, and I think it is quite possible that extra work thrown suddenly on the damaged liver, as would be the case after ligaturing a fairly large accessory duct, might cause a total suppression.

As to the second question, probably the open duct gradually closes as a result of the cicatricial changes in the operated area, and the bile may subsequently find its way round to other parts of the liver, or possibly this part of the liver undergoes cirrhotic changes.

Though quite a large number of injuries to the bile-ducts have been collected and reported by various writers, notably Eisendrath and Eliott, these probably only represent a small fraction of the total number of injuries that have been inflicted, for surgeons are ashamed of committing such errors, and rightly so, and do not feel disposed to advertise them. So long as surgeons continue to clamp and ligature structures in this region *en masse*, catastrophes are certain to occur. It is bad artistry, and can only be avoided by seeing everything, and being familiar with all the abnormalities.

I wish to express my thanks to Dr. M. J. Stewart, Professor of Pathology at the Leeds University, for his courtesy in assisting me to obtain the specimens for dissection.

REFERENCE.

[1] EISENDRATH, *Surg. Gynecol. and Obst.*, 1920, July.

SOME OBSERVATIONS ON THE TREATMENT OF ACUTE APPENDICITIS.

By R. J. McNEILL LOVE, London.

The object of this paper is to endeavour to elucidate two points in particular with regard to acute appendicitis: (I) *The best time to operate*; (II) *The best methods of drainage* when such is required. In order to acquire sufficient data from which to draw reliable deductions, I have reviewed the cases admitted to the London Hospital between 1919 and 1922—a total of 1503.

I. THE BEST TIME FOR OPERATION.

All surgeons agree that within the first twenty-four hours of the onset of acute appendicitis, the sooner the appendix is removed the better. In the majority of these cases hyperæsthesia of a band of skin above Poupart's ligament can be obtained, indicating that the peritoneum surrounding the appendix is stretched by œdema or distention of that organ. This hyperæsthesia indicates that the inflammation is limited to the appendix, and removal of that organ will abruptly terminate possible developments. Operation at this stage usually allows complete closure of the wound without drainage, and is relatively safe, as shown in *Table* 1 :—

Table 1.—Cases Operated on Within 24 Hours of Onset.

No. of Cases	Closed without Drainage	Local Drainage	Pelvis Drained	Fatal Complications	Mortality	Stay in Hospital
176	121	38	17	Secondary abscess and general peritonitis, 1	0·57 per cent	16·4 days

As treatment of cases seen within the first twenty-four hours is not a subject of controversy I shall not allude to them, and further figures only refer to cases of longer standing. However, I would suggest that the presence of hyperæsthesia is a safer guide to the condition of the appendix than arbitrary length of time, e.g., twenty-four hours.

With regard to these later cases, it has been the custom of recent years to operate as soon as the diagnosis of appendicitis was reasonably assured, with a view to removing the offending organ, preventing further infection, and draining inflammatory exudates. The mortality of operating at once is somewhere about 5 per cent. Grey Turner,[1] in a series of 681 cases, found it to be 5·13 per cent ; 1000 cases collected by Hugh Lett[2] at the London Hospital in 1912 showed a mortality of 4·3 per cent. However, these series include cases of only twenty-four hours' duration, and hence the mortality of later cases must be somewhat higher than the figures quoted. In the present series of cases operated on immediately (excluding those in *Table* 1), the mortality was 6·24 per cent.

It is becoming more recognized that the peritoneum is willing and able to deal with a considerable amount of inflammatory exudates, and therefore many surgeons now delay operation and carefully await developments. This allows the acute inflammatory reaction to subside, in which case appendicectomy is performed after an interval of about a week.

Although, in these more enlightened days of surgery, expectant treatment of a surgical condition does not appeal to surgical instincts, yet it may be remembered that, before

surgical interference became the rule, patients treated on these expectant lines usually recovered. In 1890 Sahli collected 6740 cases of perityphlitis which were treated expectantly, the mortality being 591, or only 8·8 per cent, and McBurney[3] states that 99 per cent of cases of perityphlitis are due to inflammation of the appendix. These figures are all the more striking because the importance of Fowler's position and the dietetic restrictions was not then appreciated, and purgatives, etc., were commonly misused.

When patients are given the benefit of delay, the following routine is rigidly adhered to. The patient is placed in Fowler's position, water only is allowed by the mouth, and fomentations may be applied to relieve pain. After twenty-four hours, if the signs and symptoms of infection are abating, this treatment is continued until the temperature and pulse are normal; the patient is allowed fluid diet (milk, Benger's food, etc.) when he expresses a wish for it, which time usually coincides with the approach of temperature to normal. Operation is performed one week later unless physical signs still suggest an abscess, which usually absorbs within another week. As Edmund Owen[4] pointed out, one must consider both general and local signs of infection. If, after a period of delay, the temperature and pulse remain elevated, or an abscess increases in size, then operation should be performed.

In 228 cases in which operation was delayed, 151 (66·8 per cent) responded to expectant treatment; the majority of the 77 who failed to respond were doubtless subjected to energetic treatment before arriving at hospital, which told against their settling down after admission. This suggests the criticism that even if some cases are tided over until infection subsides, the remainder who must be operated on during acute infection will show a higher mortality due to the delay, which may more than counterbalance the advantage gained in the case of the former. However, statistics fail to bear this out, and, even in cases where an operation was performed of necessity at the end of twenty-four hours or later, the mortality is approximately the same as in cases operated upon arrival in hospital. The twenty-four hours' delay, under appropriate treatment as outlined above, seems to be compensated for by the fact that it allows the patient to regain his mental calmness and settle down after his journey to the hospital, which may be sufficiently tedious to exhaust a patient who is acutely ill.

It is interesting to note that all statistics show that operation on the third day carries a much higher mortality than either earlier or later cases. Owen[4] quotes 708 cases which were operated on the third day, with a mortality of 10·7 per cent; in this series of cases the mortality was 9·8 per cent. This may be due to the fact that the resistance of the patient is at its lowest ebb at this period, and that manipulations during operation flood the tissues with toxins before the formation of an adequate quantity of antibodies.

The following tables compare the results of delayed operation with those performed as an emergency, i.e., as soon after arrival at hospital as expedient.

Table 2.—Cases in which Operation was Successfully Delayed.

No. of Cases	Days in Hospital before Operation	Drainage	Incidental Complications	Fatal Complications	Mortality	Stay in Hospital
151	8·6	Local - 54 Pelvic - 7 Total 61 = 40·4 %	Intestinal obstruction - 2 Fæcal fistula - 1 Secondary abscess - 1 Phlebitis - 2 Total 6 = 3·3 %	General peritonitis - 2 Pulmonary embolism - 1 Total 3	1·9 per cent	25·1 days

Table 3.—Cases in which Operation became Necessary because Infection did not Subside, that is, Unsuccessfully Delayed.

No. of Cases	Days in Hospital before Operation	Drainage	Incidental Complications	Fatal Complications	Mortality	Stay in Hospital
77	2·8	Local - 42 Pelvic - 31 Total 73 = 94·9 %	Intestinal obstruction - 1 Fæcal fistula 3 Secondary abscess - 3 Phlebitis - 2 Subdiaphragmatic abscess 1 Total 10 = 12·9 %	General peritonitis - 2 Ileus - - 1 Intestinal obstruction 1 Secondary abscess - 1 Total 5	6·5 per cent	29·4 days

Table 4.—Summary of all Cases treated on Delayed Lines, that is, Combining *Tables* 2 and 3.

No. of Cases	Drainage	Incidental Complications	Fatal Complications	Mortality	Stay in Hospital
228	Local - 96 Pelvic - 38 Total 134 = 58·6 per cent	7·0 per cent	8	3·5 per cent	26·5 days

Table 5.—Summary of Cases in which an Emergency Operation was Performed (*a combination of Tables subsequently analysed*).

No. of Cases	Drainage	Incidental Complications	Fatal Complications	Mortality	Stay in Hospital
1109	1048 = 94·5 per cent	13·2 per cent	67	6·24 per cent	29·1 days

The above tables suggest that in the majority of cases of acute appendicitis the infection subsides under suitable régime, and an operation in the quiescent stage can then be performed with the following advantages :—

1. Diminished immediate mortality.

2. Diminished risk of immediate post-operative complications (intestinal obstruction, secondary abscess, etc.).

3. Diminished necessity of drainage with its attendant risks, both immediate (secondary hæmorrhage, fæcal fistula, etc.), and remote (adhesions, incisional hernia, etc.).

4. Simplicity. Appendicectomy during the quiescent state is much simpler than searching for an appendix partially buried in the wall of an abscess cavity or surrounded by turgid, friable gut and congested omentum.

5. When the wound can be closed, the patient is spared the discomfort of removal of tubes, repeated dressings, etc., with a corresponding economy in hospital expenditure.

6. Length of stay in hospital is considerably diminished, and the patient is discharged with a healed wound instead of, as frequently happens, a granulating scar. Hence the period of convalescence is shortened.

One minor disadvantage of the delayed treatment is that the patient, after being successfully tided over the acute infected stage, feels restored to health and refuses the subsequent operation. This happened in one case, and fifteen months later the patient returned with a fatal recurrence. Appendicectomy must of course always be insisted on, and it is estimated that 80 per cent of cases of acute appendicitis relapse within two years unless the appendix has been removed.[5]

It would, of course, be a dangerous policy to suggest to the general practitioner and public that cases of appendicitis are not in urgent need of hospital treatment. This is undeniable, but the treatment should be expectant rather than operative. Cases should still be sent to hospital at the earliest possible time, preferably while still exhibiting the band of hyperæsthesia indicative of an intact appendix. If this is lost, the patient should be treated accordingly, and the progress closely watched so that operation can be performed at any period if necessary. Attention was drawn to the clinical significance of this cutaneous hyperæsthesia as long ago as 1903 by James Sherren,[6] and two years later the same surgeon advocated expectant treatment as described above.[7]

II. THE BEST METHODS OF DRAINAGE.

In considering this all-important question it is necessary to bear in mind the four principles involved in efficient drainage: (1) This should be as dependent as possible; (2) It should be obtained by the most direct route; (3) The risk of secondary hæmorrhage must be borne in mind; (4) The abdominal wall should be weakened as little as possible.

The site requiring drainage naturally depends upon the area infected, and this brings one to a short consideration of the various methods of approach to the appendix. Ignoring those cases in which the appendix lies on the left side, or in the region of the gall-bladder, three methods of approach may be employed—Battle's, the right rectus, and the gridiron.

Battle's incision, in which the sheath of the rectus is incised vertically and the muscle displaced inwards, undoubtedly gives a good exposure of the appendix area, and is useful in cases of uncertain diagnosis, especially in females, as it allows ready exploration of the pelvis. However, it appears to present the following disadvantages: (1) In cases where the infection is localized to the right iliac fossa the site is reached through the general peritoneal cavity, which is thus liable to contamination; and if drainage is made through this incision the tube passes through a zone of previously healthy peritoneum; hence there is risk of infecting the general peritoneal cavity. (2) When drainage is obtained through this incision the tube lies in the vicinity of the deep epigastric vessels, and secondary hæmorrhage has occurred from this cause. (3) There is risk of injury to the intercostal nerves, especially the lowest of the series, causing paralysis of the rectus muscle and lower part of the abdominal wall, predisposing to subsequent inguinal hernia.

The right rectus incision consists of a vertical incision over the lower part of the rectus muscle; the sheath is divided, and the muscle, with its nerve-supply, displaced outwards. This gives ample exposure for the removal of the appendix; the chronic appendix associated with gastric ulcer is frequently removed through this incision, i.e., a continuation downwards of the upper abdominal incision. Conversely, in the case of erroneous diagnosis when the appendix has been examined and the pathological lesion is in the upper abdomen, this area can be reached by enlarging the incision upwards. This incision, however, when employed in acute cases of appendicitis, again has the disadvantage of being placed too near the mid-line, and therefore, in many cases, the area of infection is reached through healthy peritoneum.

The gridiron incision associated with the name of McBurney, although first practised by Elliott, has the great advantage of causing the minimum of injury to the parietes. Also in the majority of cases it allows one to reach the area of infection without traversing the uninfected peritoneal cavity, and hence diminishes the risk of breaking down protective adhesions. Efficient exposure of the right iliac fossa can be obtained by continuing the incision inwards and splitting the rectus sheath transversely; and it is recommended, if exposure at a higher plane is necessary, that a second similar incision be made above the first, and the operation carried out through the two incisions. The disadvantage of the gridiron incision is that it is difficult to carry out any pelvic or upper abdominal procedure through the exposure obtained, and in these cases it must usually be closed and a more appropriate incision made.

Cases in which drainage is required may be considered in two groups according to whether generalized or local infection is present.

A. CASES OF GENERALIZED INFECTION.

In these cases the first essential is to place the patient in Fowler's position, so that exudates can collect in the pouch of Douglas. The toxæmic effects of pus in the peritoneal cavity are not in proportion to the quantity, but to the area of peritoneum in contact with it, and recent experimental work by Bolton[8] has again emphasized the relatively low co-efficient of absorption of the pelvic peritoneum. Hence, the appendix having been removed through a suitable incision, the question arises as to the most efficient route of drainage of the pouch of Douglas. I propose to analyse the three common routes of drainage, i.e., *Table* 6, through a gridiron incision: *Table* 7, through a right rectus (including Battle's); and *Table* 8, a suprapubic drainage with closure of the primary incision; the suprapubic incision may readily be made on a finger, protected by a thimble, inserted through the original wound. Below are statistics of cases in which the appendix was removed and the pelvis drained by these methods. I should like to emphasize the importance of the last column, which gives a moderately true indication of the progress of the wound—e.g., delayed healing from suppuration, or breaking down of the parietes.

Table 6.—DRAINAGE THROUGH GRIDIRON INCISION.

No. of Cases	Incidental Complications		Fatal Complications		Mortality	Stay in Hospital
421	Fæcal fistula	26	General peritonitis	14	8·8 per cent	30·4 days
	Secondary abscess	18	Secondary abscess	7		
	Intestinal obstruction	7	Intestinal obstruction	6		
	Phlebitis	6	Ileus	4		
	Subdiaphragmatic abscess	3	Infection of wound and exhaustion	2		
	Pleurisy	2	Empyema	1		
	Empyema	1	Subdiaphragmatic abscess	1		
	Parotitis	1	Secondary hæmorrhage	1		
	Pulmonary embolism	1	Pyelophlebitis	1		
	Total	65	Total	37		
	= 15·4 per cent					

Table 7.—Drainage through Right Rectus (Including Battle's) Incision.

No. of Cases	Incidental Complications		Fatal Complications		Mortality	Stay in Hospital
109	Fæcal fistula	3	General peritonitis	3	6·4 per cent	28·9 days
	Secondary abscess	3	Secondary abscess	2		
	Intestinal obstruction	1	Ileus	1		
	Phlebitis	1	Subdiaphragmatic abscess	1		
	Pleural effusion	1				
	Total	9	Total	7		
	= 8·2 per cent					

Table 8.—Closure of Primary Wound and Suprapubic Drainage.

No. of Cases	Incidental Complications		Fatal Complications		Mortality	Stay in Hospital
303	Intestinal obstruction	2	General peritonitis	9	5·3 per cent	26·5 days
	Secondary abscess	3	Ileus	2		
	Fæcal fistula	3	Secondary abscess	2		
	Phlebitis	2	Intestinal obstruction	1		
	Subdiaphragmatic abscess	1	Subdiaphragmatic abscess	1		
			Doubtful	1		
	Total	11	Total	16		
	= 3·6 per cent					

The most striking feature of the above figures is the far larger number of complications in *Tables* 6 and 7, than in *Table* 8. Three of the most important complications may briefly be considered.

1. *Mechanical Intestinal Obstruction.*—Combining *Tables* 6 and 7, we find that this occurred in 3·7 per cent of cases, compared with 0·9 per cent in the last group. The passage of the drainage tube through the peritoneal cavity undoubtedly generates adhesions ; hence the shorter the drainage tube the less the likelihood of their formation. It is found that the distance from the usual gridiron incision to the pouch of Douglas is two to three inches longer than by the suprapubic route ; and the length of drainage tube through a right rectus incision is longer in proportion to the exact position and length of the incision. The routes are demonstrated in the accompanying illustration (*Fig.* 440).

A second factor in the promotion of adhesions is that a tube in contact with parietal peritoneum is less likely to cause adhesions than one passing through coils of intestine. If infection is retrocæcal, or additional drainage of the right iliac fossa is indicated, this can be obtained by a stab wound in the loin.

2. *Fæcal Fistula.*—This is four times as common in *Table* 6 as in *Tables* 7 and 8 combined. Fæcal fistula may be due to sloughing of part of the cæcum, inability to bury the stump satisfactorily, pressure of the tube, all of which may be associated with distention of the cæcum due to temporary paralysis of the gut. Certain of these causes

are unavoidable whatever drainage is attempted ; but it would appear that pressure of the tube or the maintenance of infection along the track of the tube in the neighbourhood of the cæcum strongly predisposes to the formation of a fistula.

The majority of fistulæ close spontaneously within a fortnight, and in only one of these series of cases was an operation necessary for closure. However, a fæcal fistula means delayed healing and increased infection around a wound, and hence greater risk of post-operative hernia.

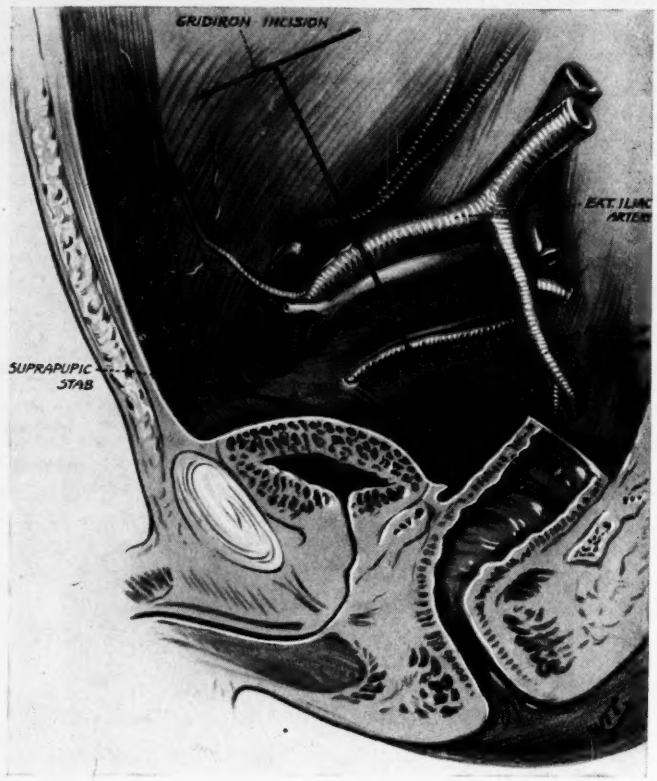


FIG. 440.—Diagram illustrating the shorter method of approach to the pouch of Douglas by the suprapubic route, compared with drainage through a gridiron incision ; also proximity of the tube through the latter incision to the external iliac vessels. As this is an antero-posterior view, the line indicating the gridiron route of drainage is foreshortened, and hence the actual distance is longer than that represented by the line.

3. *Secondary Hæmorrhage.*—The risk of secondary hæmorrhage from the external iliac vessels consequent on a drainage tube crossing the pelvic brim is well known[9] (*see Fig.* 440), and in *Table* 6 one such fatal case occurred. The external iliac artery was ligatured, but the patient died eight hours later. Secondary hæmorrhage has also been reported from the external iliac vein.[10] This danger of secondary hæmorrhage is enhanced in females, whose broader pelvis increases the obliquity of the tube, and in whom the route from the pelvis to the gridiron incision is even longer and more devious than in the male.

Some surgeons obviate the risk of secondary hæmorrhage by early removal of the tube ; but it seems difficult to foretell how long it may be necessary to retain a tube, and a premature removal predisposes to a secondary abscess.

Drainage through the right rectus incision allows the tube to enter the pelvis without dangerous proximity to these vessels, but secondary hæmorrhage has followed drainage through this incision from the deep epigastric vessels.[11]

With suprapubic drainage the tube lies in proximity to no large vessels, and hence the risk of secondary hæmorrhage is negligible. A further feature of suprapubic drainage

is that the original incision is closed, which favours its primary union, and this factor, combined with diminished complications, accounts for the shorter stay in hospital. Suppuration of the wound inevitably leads to weakening of the abdominal walls, with increased risk of post-operative hernia. Scudder and Goodall,[12] in reviewing the after-results of 640 cases, found that incisional hernia occurred in 17 per cent of cases in which the drainage tube passed through the original wound.

B. LOCALIZED ABSCESSES.

These are naturally usually located in the right iliac fossa, and therefore the gridiron incision gives direct approach. No definite surface markings are taken for the incision, but this should be made to the outer side of the swelling. In all cases where pus is suspected, it is wise to smear the superficial tissues with B.I.P.P. or a similar substance in order to diminish the risk of a superficial abscess occurring or the wounds breaking down. When the abscess is opened, care should be taken to ascertain that no pus has trickled over the brim of the pelvis, where it may be overlooked. Hence the importance of rectal examination prior to operation may be emphasized, as the collection of pus may then be recognized. Deaver[13] estimates that in 2 to 5 per cent of cases the appendix is so buried that a prolonged search for it is unjustified ; but much depends upon the skill and experience of the surgeon.

Table 9.—Drainage of Localized Infection through Wound.

No of Cases	Incidental Complications		Fatal Complications		Mortality	Stay in Hospital
249	Fæcal fistula	14	Secondary abscess	3	3·6 per cent	24·1 days
	Secondary abscess	7	Intestinal obstruction	2		
	Intestinal obstruction	3	General peritonitis	2		
	Phlebitis	2	Ileus	1		
	Subdiaphragmatic pleurisy	1	Pyelophlebitis	1		
	Total	27	Total	9		
	= 10·8 per cent					

Table 10.—Drainage of Local Infection through Stab Wound in Loin.

No. of Cases	Incidental Complications		Fatal Complications	Mortality	Stay in Hospital
27	Fæcal fistula (through gridiron incision)	1	Nil	Nil	20·9 days
	Phlebitis	1			
	Total	2			
	= 7·8 per cent				

With regard to the question of drainage of an abscess localized in the right iliac fossa, a stab wound in the loin gives the shortest and most dependent route, especially when the appendix is retrocæcal. The stab wound can readily be made on the points of a pair of

sinus forceps introduced through the wound, care being taken not to injure a prolapsed kidney if such a condition co-exists. The advantages of drainage through a stab wound in the flank are suggested by the figures in *Table* 10; although unfortunately the number of cases is rather small, their shorter stay in hospital is noteworthy.

SUMMARY.

1. All cases of acute appendicitis should be operated on immediately if a band of hyperæsthesia is still present.

2. Under appropriate treatment the majority of remaining cases subside, and the appendix may be removed seven to ten days after the temperature and pulse become normal. As compared with emergency operation this line of treatment shows a lower mortality, fewer complications, and a shorter stay in hospital.

3. In cases in which expectant treatment fails, twenty-four hours' delay does not appear to influence the prognosis adversely.

4. Except in cases where difficulty is anticipated (e.g., recurrent cases), or where the diagnosis is doubtful, the gridiron is the incision of choice, as manipulations can then be limited to the infected area. In other cases a right rectus incision, displacing the muscle outwards, is preferable to Battle's, there being less risk of injury to the nerves.

5. Stab drainage, suprapubic or in the flank, favours primary union of the original wound, thus diminishing the length of stay in hospital and risk of subsequent incisional hernia. Also adhesions and fæcal fistulæ are less likely to develop, and the risk of secondary hæmorrhage is obviated.

I am indebted to the various surgeons at the London Hospital for permission to include in these series cases which were under their observation and treatment.

REFERENCES.

[1] TURNER, *Brit. Med. Jour.*, 1913, March 8.
[2] LETT, *Short's Index of Prognosis*, Wright, Bristol.
[3] MCBURNEY, *Trans. Med. Soc. New York*, 1891, Feb.
[4] OWEN, *Appendicitis: A Plea for Early Operation*, 1914, Wright, Bristol.
[5] *Ann. of Surg.*, 1910, ii, 801.
[6] SHERREN, *Lancet*, 1903, Sept. 15.
[7] SHERREN, *Practitioner*, 1905, June.
[8] BOLTON, *Jour. Pathol. and Bacteriol.*, 1910, Oct.
[9] RAMSEY, *Lancet*, 1903, i, 1590.
[10] O'CONNOR, *Ibid.*, 1902, Aug.
[11] *Practitioner*, 1920, July.
[12] SCUDDER AND GOODALL, *Boston Med. and Surg. Jour.*, 1911, July 6.
[13] DEAVER, *Abdominal Surgery.*

A NOTE ON THE OPERATION FOR THE RADICAL CURE OF FEMORAL HERNIA.

By ERNEST W HEY GROVES, Bristol.

About fifteen years ago, when I had to operate upon a strangulated femoral hernia in a stout woman, I divided Poupart's ligament in order to obtain a better view of the neck of the sac and its contents. It was a case in which the viability of the gut was in serious question ; I feared that a piece of bowel higher than I had access to from the groin might be damaged, and I could not pull down further intestine without fear of rupture. Therefore I had recourse to the expedient of dividing Poupart's ligament with Gimbernat's ligament close to their attachment to the pubic spine. This gave an exposure so excellent that full investigation was possible, and, after dealing with the sac and its contents, I was able to attach the conjoined tendon to Cooper's ligament with much greater facility than if this had had to be done under the overhanging tendon of the external oblique.

It occurred to me on the occasion of this operation that, quite apart from dealing with strangulated bowel, this procedure afforded greater precision in performing a radical cure than any other operation I had hitherto seen or performed.

I have therefore carried out this femoro-inguinal operation as a routine ever since. I think it is probable that many other surgeons have done the same thing ; but I have read no description of this operation, whilst visitors who have watched it have been interested in it as a new procedure.

I therefore venture to describe it in detail and to give the results in a consecutive series of cases.

The hernia is exposed by a vertical incision about four inches long, the middle of which corresponds with the fold of the groin. A very little dissection then reveals the hernial sac, lying on the pectineus muscle, with Poupart's ligament crossing its neck, and on its outer side the femoral vein joined by the long saphenous vein (*Fig.* 441).

The attachment of Poupart's ligament to the pubic spine is then cut close to the bone and the ligament turned outwards (*Fig.* 442). This gives a clear view of the neck of the sac, which is then opened and its contents dealt with according to their condition. Should resection of the bowel be necessary, this can be done perfectly well by the exposure thus afforded. When the sac has been cut off and its neck ligatured, the femoral ring and canal can be obliterated (*Fig.* 443). The conjoined tendon is defined and sewn down to Cooper's ligament and the upper edge of the pectineal fascia. The outer one or two stitches also take up the femoral sheath on the inner side of the femoral vein, so as completely to close the abdominal cavity from the thigh. Poupart's ligament is then laid over the line of suture between the conjoined tendon and Cooper's ligament, beginning from the vein and working inwards towards the spine of the pubis (*Fig.* 444). As the inner attachment of Poupart's ligament has been divided, there is no tension in this structure and it can be made to follow the line of the pubic bone, and the final attachment of its cut end will be about ½ in. further outwards than it was originally. In my later cases, instead of cutting Poupart's ligament, I have chipped off a small part of the pubic spine (*Fig.* 445), and at the final stage of the operation the detached piece of bone is fixed by means of a single ivory nail.

The advantages which this combined femoral and inguinal operation afford would seem to me to be as follows :—

1. In cases of strangulated hernia it gives ample room to deal with damaged bowel and, if necessary, to do a resection.

2. It gives all the advantages of the inguinal approach, i.e., the possibility of closing the femoral canal from above, without the necessity of trying to drag up the hernial sac through the femoral canal.

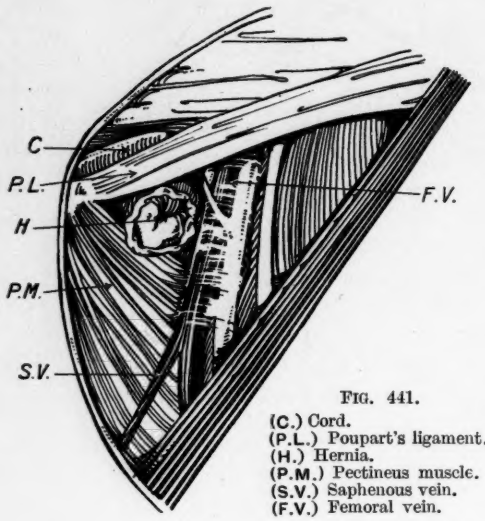


FIG. 441.
(C.) Cord.
(P.L.) Poupart's ligament.
(H.) Hernia.
(P.M.) Pectineus muscle.
(S.V.) Saphenous vein.
(F.V.) Femoral vein.

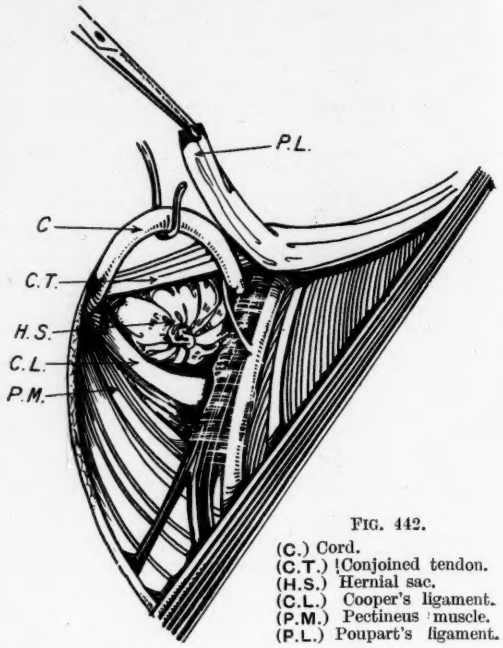


FIG. 442.
(C.) Cord.
(C.T.) Conjoined tendon.
(H.S.) Hernial sac.
(C.L.) Cooper's ligament.
(P.M.) Pectineus muscle.
(P.L.) Poupart's ligament.

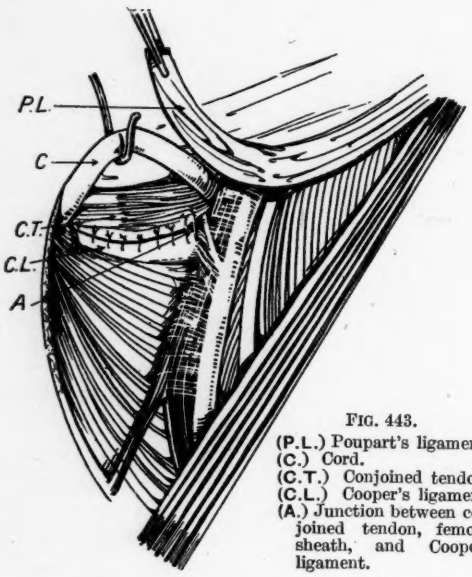


FIG. 443.
(P.L.) Poupart's ligament.
(C.) Cord.
(C.T.) Conjoined tendon.
(C.L.) Cooper's ligament.
(A.) Junction between conjoined tendon, femoral sheath, and Cooper's ligament.

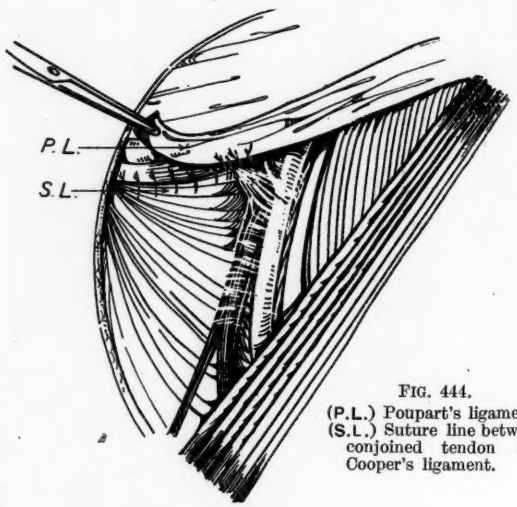


FIG. 444.
(P.L.) Poupart's ligament.
(S.L.) Suture line between conjoined tendon and Cooper's ligament.

3. It allows the suture of the conjoined tendon to Cooper's ligament to be undertaken with great precision, unhindered by the overlying Poupart's ligament.

4. Poupart's ligament, being freed from tension, can be snugly sutured as an extra covering over the line of suture between the conjoined tendon and Cooper's ligament.

Results of the Operation.—I have looked up all cases operated upon by myself during the period 1912 to 1922, and I have been able to see and examine 22. This

is of course a small number, but sufficient to give some criterion of the permanency of cure.

In one case only was the complete radical operation not attempted, this being on account of the gangrenous condition of the large omental contents. It was intended to complete the radical operation at a later date, but the patient did not care to have this further operation, and she still has a hernia about the size of a duck's egg.

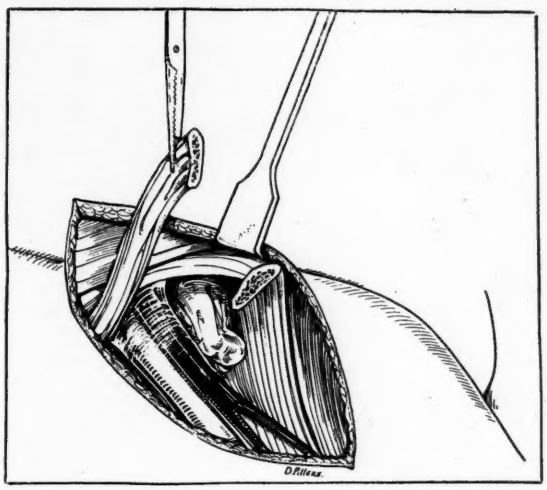


FIG. 445.—Right femoral hernia; removal of a piece of bone from the pubic spine.

All the other 21 cases have remained soundly healed and free from recurrence. The sex incidence was 21 women and 1 man. The ages varied between 23 and 73. In 9 cases the operation was necessitated by strangulation.

All the cases have been hospital patients, and they have, with one exception, been able to return to housework or work in factories after the operation. The exception is a woman, now 72 years old, who is bedridden with heart disease.

In 3 cases the patients have borne children since the operation, without any recurrence of the hernia or inconvenience from the site of the operation.

ABNORMALITIES OF FIXATION OF THE ASCENDING COLON: THE RELATION OF SYMPTOMS TO ANATOMICAL FINDINGS.

By ADAMS A. McCONNELL and T. GARRATT HARDMAN, Dublin.

SUMMARY.

1.—The Normal Ascending Colon:
   a. Anatomy.
   b. Radioscopic examination.
2.—Development and Fixation.
3.—Variations in Fixation and Resulting Types of Ascending Colon.
4.—Variations in Form of Ascending Colon; Clinical Picture.
5.—Traction of Ascending Colon on other Structures:
   a. Superior mesenteric artery.
   b. Duodenum.
   c. Gall-bladder.
   d. Pyloric end of stomach.
   e. Right kidney.
6.—Remarks on Radioscopic Technique.
7.—General Observations.
8.—General Treatment.

Stimulated by Waugh's paper on the mobile ascending colon, and realizing the possible significance of his conception in abdominal surgery, we decided to observe the ascending colon in every abdominal case, and to inquire whether its anatomical condition was in any way responsible for the symptoms or disease encountered. The result of our investigation can be presented most readily by describing what we consider the normal ascending colon, the manner of its development, the variations of the normal which we have encountered, and the symptoms associated with these variations.

1. THE NORMAL ASCENDING COLON.

Fig. 446.
Normal ascending colon.

a. **Anatomy.**—Anatomists describe this portion of the gut as it is seen in the dead subject; surgeons visualize it as it appears during a laparotomy; both see it when the subject is recumbent. Radioscopy alone reveals its position in the erect living subject.

The normal ascending colon lies in direct contact with the posterior abdominal wall, and is held in position by the reflexion of the parietal peritoneum. The hepatic flexure and the beginning of the transverse colon are similarly fixed to the anterior surface of the right kidney and to the second stage of the duodenum respectively; by means of this fixation the ascending colon is maintained in a vertical position as a practically straight tube presenting some slight curves or flexures (*Fig.* 446). Such is the conception which is obtained from observation in the cadaver.

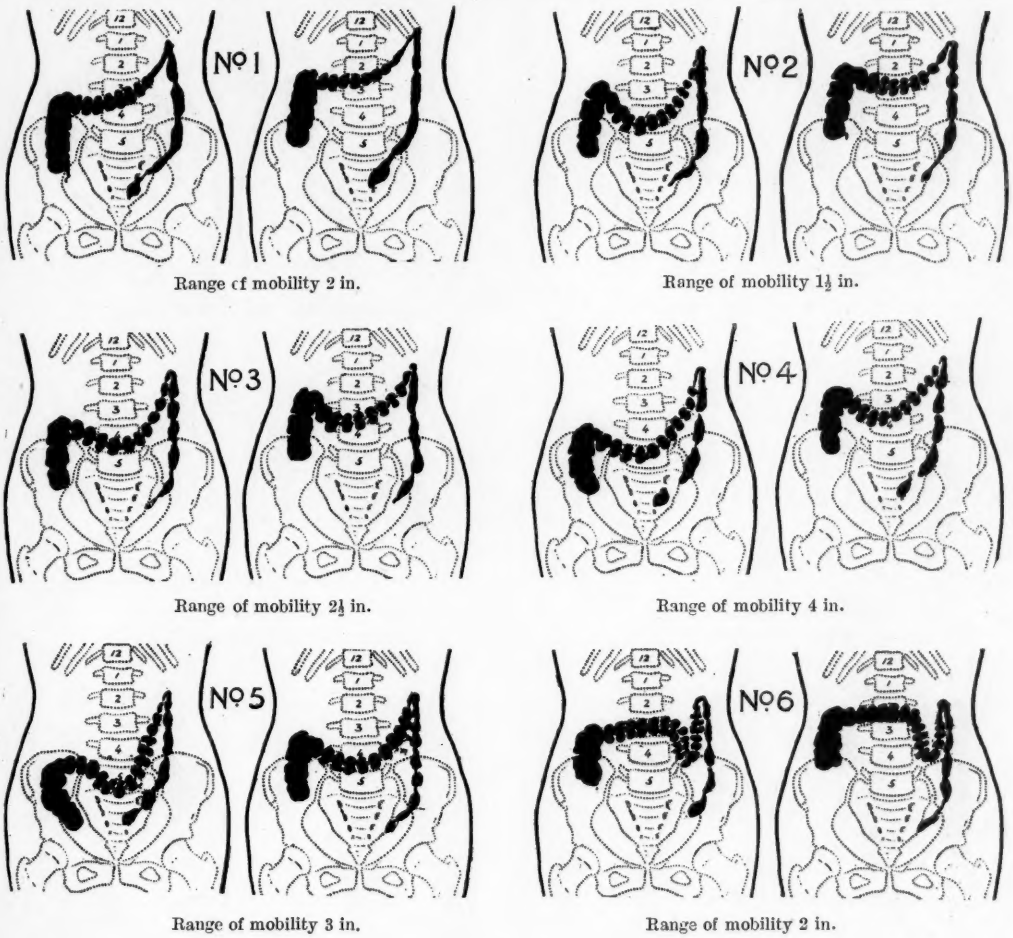
b. **Radioscopic Examination.**—This was carried out with the fluorescent screen in a series of individuals who presented no symptoms of abdominal or other disease. The results are as follows:—

Males : Ages between 21 *and* 38 (*Fig.* 447) :—

Average length, including cæcum	.. ..	7 inches.
,, position of hepatic flexure above iliac crest, *erect*	..	1½ ,,
,, ,, ,, ,, ,, ,, *recumbent*	..	4 ,,
,, range of vertical excursion	.. ..	2½ ,,

FIG. 447.—MALE COLONS. Nos. 1 to 6.

(*In each instance the diagrams on the left show patient in the erect position; those on the right in the recumbent position.*)



Range of mobility 2 in. Range of mobility 1½ in.

Range of mobility 2½ in. Range of mobility 4 in.

Range of mobility 3 in. Range of mobility 2 in.

The ascending colon formed practically a right angle with the transverse colon. The former was practically straight, and the diameter of the cæcum was but slightly greater than that of the succeeding segment of gut. There was very little lateral mobility of the ascending colon on palpation.

Females : Ages between 20 *and* 29 (*Figs* 448, 449) :—

Average length, including cæcum, *erect*	.. .. ..	7 inches.
,, ,, ,, ,, *recumbent*	.. .. ..	8 ,,
(*the longest was* 9 *in., the shortest* 6½ *in.—recumbent.*)		
Average position of hepatic flexure above iliac crest, *erect*	..	¼ ,,
,, ,, ,, ,, ,, ,, *recumbent*	..	3 ,,
,, range of vertical excursion	.. ..	2¾ ,,

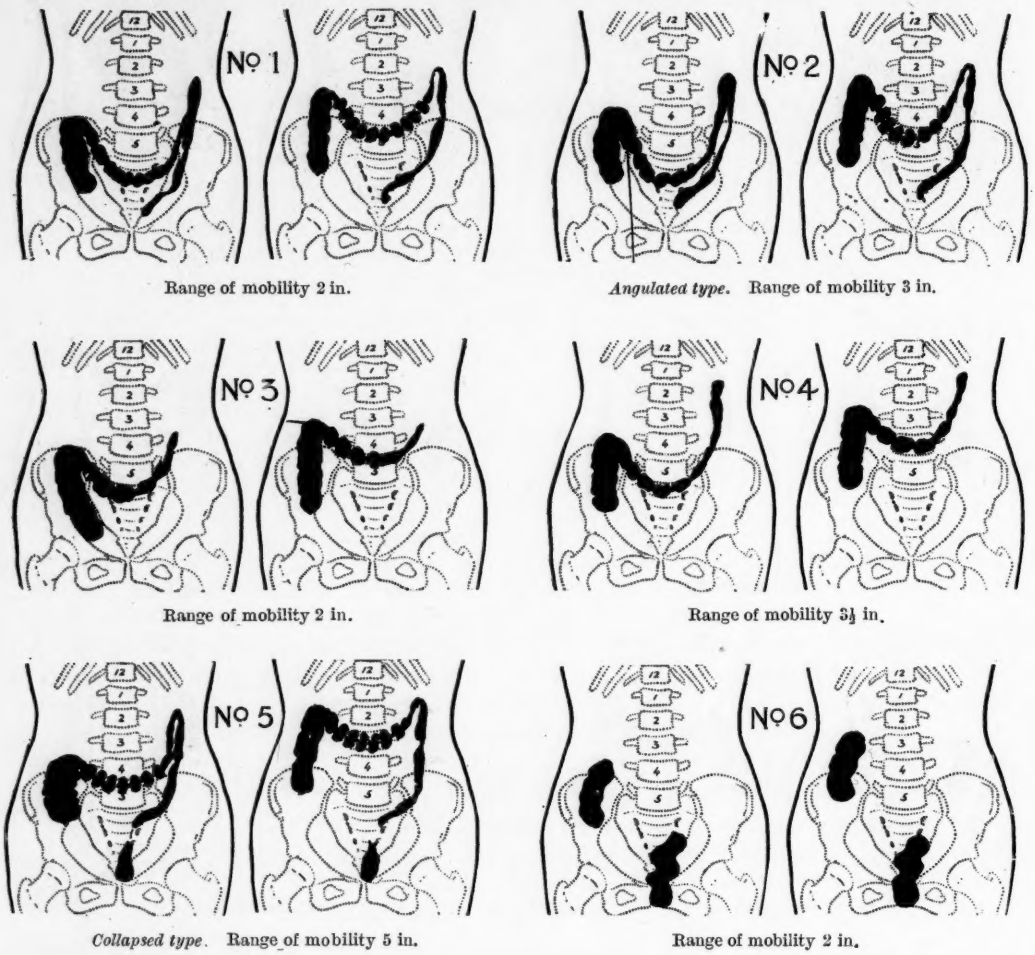
In 82 per cent the ascending colon was straight. In 18 per cent it presented one or more flexures along its course. The angle between the ascending and transverse portions

of the colon was less than a right angle. The diameter of the cæcum was practically the same as that of the ascending colon. There was but slight lateral mobility on palpation in 92 per cent.

In determining the length of the ascending colon by radioscopy one must be careful, in making the measurement, that the summit of the barium shadow actually coincides

FIG. 448.—FEMALE COLONS. Nos. 1 to 6.

(In each instance the diagrams on the left show patient in the erect position; those on the right in the recumbent position.)



Range of mobility 2 in. *Angulated type.* Range of mobility 3 in.

Range of mobility 2 in. Range of mobility 3½ in.

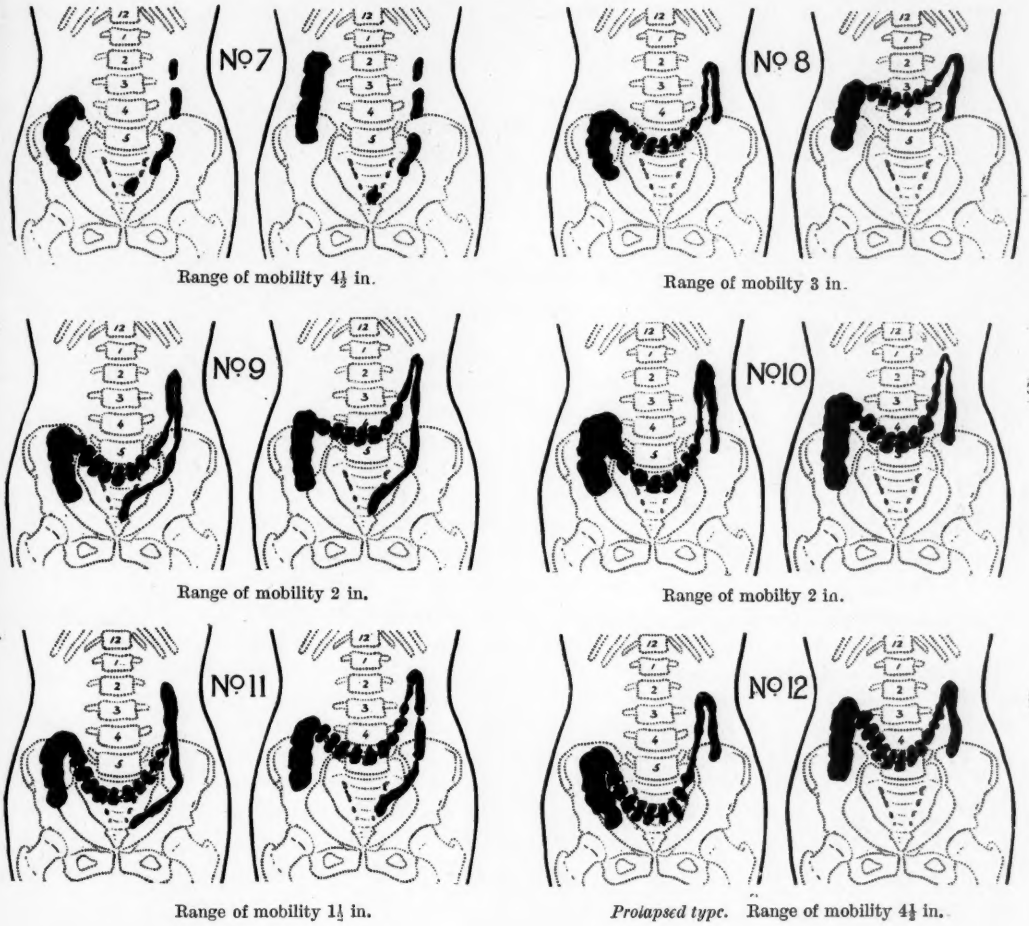
Collapsed type. Range of mobility 5 in. Range of mobility 2 in.

with the top of the hepatic flexure. This portion of the gut, if empty, will (it is hardly necessary to point out) cast no shadow, and hence the summit of the opaque content might be erroneously taken as the top of the hepatic flexure; but if there is an accumulation of gas in the flexure the bowel will be clearly outlined and there is no danger of such an error. We have found that the best time in which to make measurements is twenty-four hours after the ingestion of the meal, when, as a rule, the transverse colon as well as the cæco-ascendens is rendered visible by its opaque contents. These measurements we regard to be of the first importance in ascertaining the degree of fixation or lack of fixation of the hepatic flexure. In pronounced cases of what we may refer to as *mobile colon*, the position of the flexure varies enormously with the posture of the patient, or, in other words, flexion occurs at two different points in the colon, according as the patient is erect or recumbent. From our series of normal individuals we have conceived—whether rightly or wrongly—that the average normal ascending colon is practically constant in length in

the erect and recumbent postures, although its position may vary to the extent of several inches; but any considerable variation in length produced by change of posture indicates a loss of normal fixation of the hepatic flexure.

These observations show that considerable differences exist in the ascending colon, not only in the two sexes but in individuals of the same sex. Radioscopic examination

FIG. 449.—FEMALE COLONS—*continued.* Nos. 7 to 12.
(*In each instance the diagrams on the left show patient in the erect position; those on the right in the recumbent position.*)



Range of mobility 4½ in. Range of mobilty 3 in.

Range of mobility 2 in. Range of mobilty 2 in.

Range of mobility 1½ in. *Prolapsed type.* Range of mobility 4½ in.

of a large number of individuals of middle age who have never had abdominal symptoms would do much to elucidate the anatomy of the ascending colon in the living. The type of ascending colon which in our experience was never associated with the symptoms described below was that present in 66·6 per cent of symptomless males. Its characteristics are as follows:—

Length	7 inches.
Position of hepatic flexure above iliac crest—*erect*	2 inches.
" " " " " *recumbent*	4 inches.

The angle between the ascending and transverse colon is nearly a right angle. The cæcum has practically the same diameter as the ascending colon. There is no lateral mobility. It is very unusual to see a range of vertical mobility of the hepatic flexure of less than 2 in.

We do not hold that all types which differ from this are abnormal, but we suggest that this is the type to which evolution tends.

2. DEVELOPMENT AND FIXATION OF THE ASCENDING COLON.

About the time of birth the cæcum is situated in front of the right kidney, near the gall-bladder, and the proximal part of the colon possesses a complete mesentery. Then both the cæcum and ascending colon gradually descend towards the right iliac fossa. Adhesions form between the parietal peritoneum and the posterior aspect of the ascending colon and its mesentery. These two adherent peritoneal surfaces disappear, so that the colic vessels and the colon itself become retroperitoneal, and the normal adult condition is reached. An ascending mesocolon is as abnormal as a cleft palate.

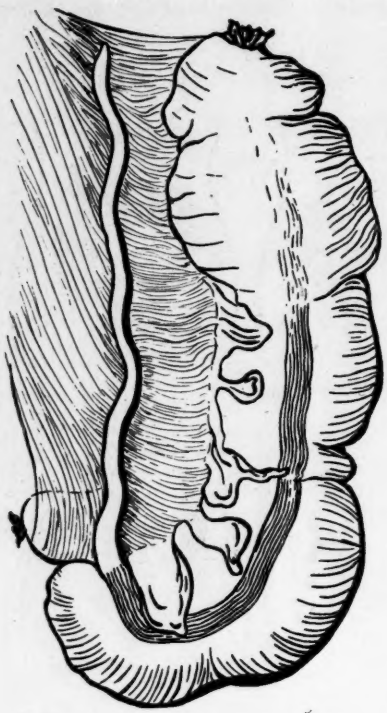


FIG. 450.—Primitive mesentery entire.

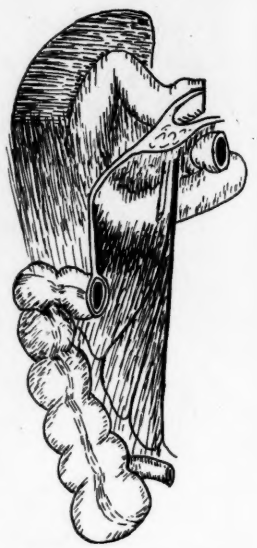


FIG. 451.—Prolapsed type.

3. VARIATIONS IN FIXATION AND RESULTING TYPES.

The variations of this normal process which we have met surgically are :—

a. The new adhesions may persist in excess to the lateral aspect of the ascending colon and constitute varying degrees of parieto-colic folds. We have observed these folds so frequently in newborn children and infants under two years in the anatomical departments of Trinity College, Dublin, and the Royal College of Surgeons, that we regard them as congenital.

b. The new adhesions may not form at all, or may remain too attenuated to function, so that the primitive mesentery persists in its entirety (*Fig.* 450), when one of two things may happen : (i) The whole ascending colon sinks to the limit permitted by its mesentery, and remains straight—the *prolapsed* type (*Fig.* 451). This type is usually associated with lax abdominal muscles. (ii) The cæcum may be prevented from sinking, and the hepatic flexure approximates to the cæcum, so that the ascending colon resembles a collapsed concertina—the *collapsed* type (*Fig.* 452) ; or it becomes sharply bent at one point—the *angulated* type (*Fig.* 453).

FIG. 452.—Collapsed type.

FIG. 453.—Angulated type of ascending colon.

c. The new adhesions may fix only the lower or middle portion of the ascending colon, and the upper part may remain free, so that the hepatic flexure tends to fall down in front of or to the side of the lower portion, and produce a sharp angulation of the gut —the *angulated* type : thus this type may or may not have an attachment to the parietes.

X-ray Appearances.—Radioscopic examination with the fluorescent screen shows that the prolapsed type of ascending colon remains practically straight both in the erect and recumbent positions (*Fig.* 449, *No.* 12, and *Figs.* 454 and 455). Its length is very little

FIG. 454.—Twenty-four hours after meal.—*Erect.*

FIG. 454A.—The same case.—*Recumbent.*

altered. The vertical excursion of the hepatic flexure may be as much as 6 in. Its distinctive feature is its lateral mobility—one can push it to the middle line or beyond; sometimes it might be termed a 'floating' colon, so pronounced is its mobility in every direction. The collapsed and angulated types appear normal with the patient recumbent; when the erect position is assumed, however, the appearance of both is that of an irregular mass in the right iliac fossa (*Figs.* 456, 457, 458). Palpation under the screen reveals the difference in the two types (*Fig.* 449, *Nos.* 2 and 5, and *Fig.* 459). We have encountered the prolapsed type in 58 per cent, the collapsed in 3 per cent, and the angulated in 31 per cent, whilst 8 per cent were apparently normal on *x*-ray examination.

FIG. 455.—Twenty-four hours after meal.—*Erect.*

The type of ascending colon present is therefore a function of its fixation, modified in some degree by the tonicity or atony of the musculature of the anterior abdominal wall. When the ascending colon has no attachment to the posterior abdominal wall, it is supported altogether by its attachments to the right kidney and second stage of the duodenum, by its own mesentery, and, through the proximal part of the transverse colon, by the gastrocolic omentum and pyloric end of the stomach. The results of faulty fixation fall, therefore, into two general classes, namely: (1) *Variation in the actual form of the ascending colon*—the angulated and

collapsed types ; and (2) *Excessive traction on the structures to which the ascending colon is attached,* varying inversely with the degree of attachment to the posterior abdominal wall and with the tonicity of the abdominal musculature.

FIG. 456.—Forty-eight hours after meal.—*Angulated type, erect.*

FIG. 456A.—The same case.—*Recumbent.*

4. VARIATIONS IN THE FORM OF THE ASCENDING COLON.

Mass Movement in a Normally Fixed Colon.—The contents of the colon are semi-solid, and are propelled onwards, not by short peristaltic waves occurring frequently, but by very infrequent vigorous contractions which appear to start in the cæcum or proximal end of the ascending colon, and travel over a considerable distance, driving the contents

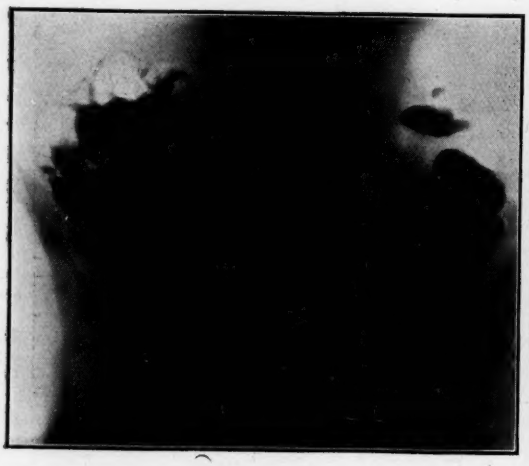


FIG. 457.—Forty-eight hours after meal.—*Erect.*

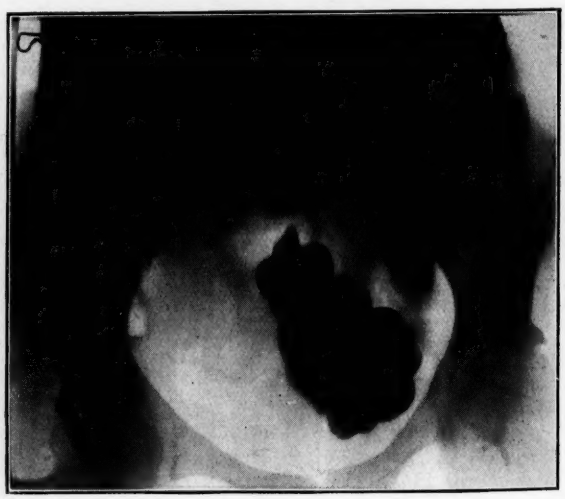


FIG. 457A.—The same case.—*Recumbent.*

of the bowel before them in one mass or column. At the moment when the contractions start, a remarkable change occurs in the transverse colon. The normal haustration disappears, and the bowel, which a moment before had formed a sagging loop between two fixed points, appears to shorten and to become like a rigid tube straight across the abdomen. This shortening of the transverse colon is due presumably to contraction of

the longitudinal muscular bands. The whole contents move rapidly for a distance of perhaps 6 to 12 in., and the movement stops as suddenly as it commenced, the haustral contractions reappearing and the colon returning to its normal position and quiescence.

FIG. 458.—*Erect.*

FIG. 458A.—The same case.—*Recumbent.*

These movements are only very rarely seen, and have been observed by one of us on not more than three occasions in the course of hundreds of examinations. The result of this contraction is that, no matter how acutely angulated the hepatic flexure may be, it assumes a right-angled bend during contraction.

FIG. 459.—*Erect.*

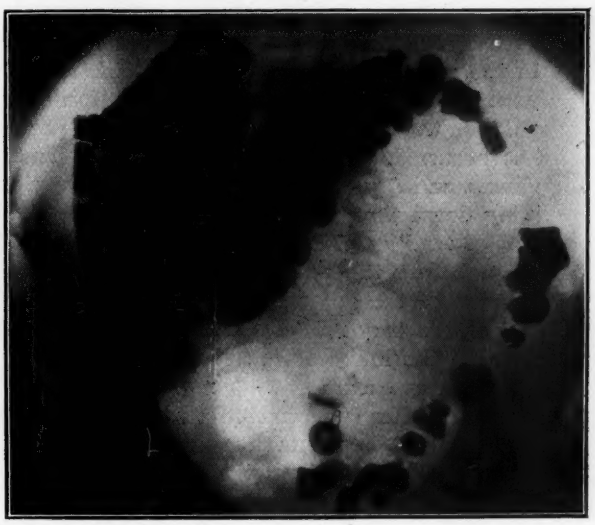


FIG. 459A.—The same case.—*Recumbent*

Mass Movement in the Imperfectly Fixed Colon.—We have never been fortunate enough to observe this movement in the deformed or mobile type of ascending colon ; it seems probable, however, that, when the hepatic flexure is not fixed, both the ascending and the transverse segments lose a point of purchase, and complete mass movement does

not occur, but a small portion of the colonic contents passes along the gut at each attempt. The presence of a strong parietocolic fold halfway up the ascending colon may afford a fixed point, and the mass movement takes place ; but probably not so perfectly as when the hepatic flexure itself is fixed.

It is in cases of the collapsed and angulated types of ascending colon that most cæcal distention is observed. In these the contents of the cæcum have to be forced past a kink or kinks and against the dead weight of the superimposed colon. In the absence of a point of fixation, contraction of the colonic musculature does not obliterate these kinks, but exaggerates them so that they constitute a true obstruction, resulting in dilatation of the cæcum and the proximal part of the ascending colon.

When the individual assumes the recumbent posture the hepatic flexure falls into the normal position, the kink or kinks disappear, and the obstruction is relieved. This periodic relief by recumbency may account in part for the long period that often elapses before these congenital defects produce clinical evidence of their presence.

The Relation of Symptoms to the Angulated and Collapsed Ascending Colon.—

Case 1.—Female, age 30. About six months before admission she began to suffer from loss of appetite, and discomfort in the right iliac fossa after food. All kinds of food disagreed with her, and her diet became restricted to small quantities of tea and milk. After a few weeks the discomfort became actual pain ; it came on during breakfast and immediately after other meals, and was accompanied by nausea. After she had taken a few mouthfuls at breakfast she felt so sick that she could not eat any more. In the course of an hour or two she resumed the interrupted meal. When she stayed in bed for breakfast she experienced neither pain nor nausea, and had less discomfort after other meals. If she remained in bed all the time she could take food freely. Never vomited. Was never constipated until these symptoms appeared. Since their onset she became thinner and somewhat anæmic. Tenderness was present in the right iliac fossa over a distended cæcum. Radioscopy demonstrated the angulated type of ascending colon in the erect posture. It straightened out when the patient lay down. Excursion of hepatic flexure 4 in. A large amount of the meal remained in the proximal colon after thirty-six hours. The rest of the gastro-intestinal tract appeared normal.

Operation.—Upper two-thirds of ascending colon possessed complete mesentery. Parietocolic fold fixed lower one-third. Stomach and duodenum normal. Appendix normal. Appendicectomy—colopexy. Result after one year and nine months—'quite well'.

When the symptoms were referred to the right iliac fossa, the ascending colon was of the angulated or collapsed type. The ascending colon usually has its heaviest load in the morning. When the patient gets up, the angulation or collapse becomes marked ; when he takes his breakfast, contraction of the colon begins and he suffers from colicky pains in the right iliac fossa or the lower part of the abdomen. If dilatation of the cæcum has occurred, the patient experiences a sensation of sinking or fullness, accompanied by nausea and dry eructations. If he lies down the pain is relieved ; if he has breakfast when lying down the pain is prevented : hence the statement, " I cannot do without my early cup of tea." Colonic contraction stimulated by ingestion of food empties the ascending colon before the patient assumes the erect posture, and therefore before angulation or collapse of the gut takes place. If he goes to stool after breakfast in bed, he has a free and satisfactory motion ; if he has got up for breakfast, the evacuation is but of the contents of the lower bowel, and he suffers from discomfort in the right iliac fossa until the bowels move again. This morning discomfort may be repeated, or will ultimately be repeated, after every meal. If the normal evacuation of the bowels takes place after the evening meal, it is sometimes during that meal that pain has its onset or exaggeration. Once these symptoms appear, they usually continue daily until the patient learns how to control them. Sometimes, however, he suffers from attacks at irregular intervals.

These symptoms have no marked relation to constipation ; often indeed the patient will not admit that he is constipated. Radioscopy, however, frequently demonstrates some degree of stasis in the proximal colon ; the more pronounced the stasis, the more does the patient suffer from general symptoms of toxæmia—persistent headache, nausea, loss of appetite, lassitude, and wasting. As time goes on he becomes anæmic and nervous.

There is a definite relation in some cases between physical or mental exhaustion and the onset or recurrence of these attacks. Some of the patients developed symptoms only

when tired. Apparently colonic tone was sufficient to overcome whatever colonic obstruction was present, until fatigue removed the former and left the latter unchanged. The typical symptoms, in brief, are those of subacute obstruction in the ascending colon—discomfort, pain, or a sinking sensation in the right iliac fossa coming on during or immediately after a meal, especially breakfast, and *relieved by lying down*. Nausea is common, vomiting unusual. Sometimes adhesions have fixed the colon so kinked, and the symptoms are independent of posture. Physical examination reveals distention of the cæcum. A parietocolic fold was found in nearly every case of the angulated type operated on. Jackson's veil-like membrane was present in 15 per cent of all cases. Not every patient with an angulated or collapsed colon has these symptoms, but every patient with these symptoms has an angulated or collapsed colon.

The fact that this clinical picture was always associated with either the angulated or collapsed type of ascending colon, and not with the prolapsed straight type, has convinced us that it is only when an obstruction can be demonstrated in this segment that marked symptoms are referred to the right iliac fossa. So long as the ascending colon is straight, its mobility, though interfering with complete evacuation, does not markedly prevent it.

The complete clinical picture is preceded by less definite and suggestive symptoms. When about twenty years of age the patient becomes gradually conscious of vague discomfort in the lower part of the abdomen—a feeling of heaviness or slight nausea during or immediately after a meal. He finds that he has to go to stool twice instead of once. Ultimately he discovers that relief comes if he lies down. Palpation reveals slight tenderness in the right iliac fossa ; the distended cæcum is noted, but often ignored. Medicine does no good, and then a normal appendix is removed, which operation may mark the beginning of a surgical pilgrimage. Other patients are perfectly well for twenty or more years, when the condition of the colon is discovered in seeking for the cause of sudden acute pain in the right iliac fossa. It is always safer to make a diagnosis of acute appendicitis in doubtful cases ; but if the appendix be not diseased, the culpability of the colon should be suspected. In such acute cases we have been impressed with the apparent well-being of most of the patients. Although they may writhe with pain, the temperature is not raised, the rate of pulse is not quickened, and their appearance is not toxic.

Symptoms somewhat similar to those given as typical have been ascribed to a 'distended cæcum' apparently idiopathic, and to incompetency of the ileocæcal valve. We have not once observed the latter in these cases, but the former is constant. J. C. Roux, in a paper reviewed by Robert Hutchison in the *Medical Annual* of 1922, gives an account of what he has termed cæcal constipation. He notes the occurrence of some of the above symptoms and the relation of discomfort in the right iliac fossa to posture. Among the causes of this variety of constipation he includes a mobile ascending colon, the presence of false membranes, inflammatory pericolitis, and cæcal ectasis. All the cases in our series which had cæcal ectasis had the angulated or collapsed types of mobile colon and demonstrable obstruction. Surely dilatation of a tube makes one suspect obstruction.

Patients with the prolapsed type of ascending colon seldom referred their symptoms to the right iliac fossa ; when they did, however, the clinical picture was never so complete as in the angulated type. Vague discomfort, a sensation of heaviness, sinking, or pain after food, sometimes relieved by recumbency, marked these cases. None of them presented acute symptoms.

Eighteen patients referred all or some of their symptoms to the right iliac fossa. Of these, one had the collapsed type of colon, twelve the angulated, and five the prolapsed. Four of the cases with the angulated type were admitted as acute appendicitis. At operation no lesion was found in the appendix. Gastro-enterostomy for gastric ulcer had been performed in two cases more than a year before, with relief of epigastric symptoms, but those referred to the right iliac fossa persisted or became prominent. Two patients had had the appendix removed without any relief. Apart from the eighteen there were six cases in which the ascending colon was angulated and no symptoms were referred to the right iliac fossa.

Treatment.—Many patients have learned by experience how to relieve the symptoms, and many physicians are empirically successful. The administration of liquid paraffin, a dose of salts before breakfast in bed, and recumbency for half an hour after each meal, may keep a patient free from discomfort, and some there be who can adopt such a régime. Removal of a normal appendix does not affect the progress of the case. Cæcoplication by itself is based on a false idea of the cause of the dilatation. We do not compress the head for hydrocephalus, nor plicate a dilated ureter. Cæcoplication may be useful along with colopexy. Fixation of the cæcum alone leaves the condition unchanged when the colon is angulated or collapsed, whilst in the prolapsed type angulation or collapse is produced; an empty stocking will not stand in a shoe. Resection of the cæcum and ascending colon is unwarrantable in early cases; one does not amputate the foot because it is flat. When, however, long-standing obstruction has left the cæcum simply an inert atonic sac, an ileo-cæcal resection is rational. Division of parietocolic folds turns an angulated colon into a prolapsed one, and substitutes one set of symptoms for another. The whole ascending colon must be made straight and then fixed to the posterior abdominal wall. It must be remembered that the diagnosis of the angulated and collapsed types of ascending colon cannot always be made at operation, for then the colon may have fallen into a normal position, and one can say no more than that it is mobile. As a rule, deformity is revealed only by *x*-ray examination.

More than a year has elapsed since the operation in twelve of these cases. Eleven of the twelve report 'well'. Colopexy was performed in each. The twelfth case had, in addition to mobility of the colon, a well-marked Lane's kink and ileal stasis. The ileal band was divided and the appendix removed; the colon was not fixed. This patient's symptoms returned within ten days of leaving hospital.

5. TRACTION ON THE STRUCTURES TO WHICH THE ASCENDING COLON IS ATTACHED.

When the ascending colon fails to acquire a wide surface of apposition to the unyielding posterior abdominal wall, its suspension depends partly on its own mesentery, and on its peritoneal attachments to the renal fascia and second stage of the duodenum. Normally the proximal portion of the transverse colon is largely supported by the more fixed hepatic flexure. When the latter is mobile, it ceases to be a support, and becomes partly dependent on the beginning of the transverse colon, which, in turn, drags on the gastrocolic omentum and on the pyloric portion of the stomach. This strain on the stomach exerts itself primarily on the greater curvature, which, however, can yield to it, being essentially a mobile portion of the stomach; but the lesser curvature of the stomach is relatively fixed by the attachment of the lesser omentum, and consequently is subject to more tension.

In a considerable number of cases there are congenital peritoneal folds passing from the gall-bladder to the duodenum or to the hepatic flexure; through these the mobile ascending colon may drag indirectly or directly on the gall-bladder.

The fact that a mobile ascending colon does exert traction on the structures to which it is attached was established by noting the position of the hepatic flexure and of the ileocæcal junction under the radioscopic screen with the patient erect. At operation the hepatic flexure was placed in its predetermined position, and the effect of this replacement on the duodenum and pyloric end of the stomach observed. The ileocæcal junction was similarly replaced in order to note the effect on the superior mesenteric artery and third stage of the duodenum.

If the ascending colon be in its normal relation to the posterior abdominal wall, its position is affected little, if at all, by laxity of the anterior abdominal wall. Radioscopy and laparotomy have frequently shown a fixed colon in cases with chronic laxity of the anterior abdominal wall. Whatever be the condition of the anterior abdominal wall, the presence of a mesentery allows the ascending colon to sink to an extent not possible to the relatively fixed structures to which it is attached, and to exert a certain degree of traction on them. Strong abdominal muscles may keep the ascending colon up, but they do not keep it up all the time, nor can they keep it straight.

The effects of traction in general reach their maximum under the following conditions: (i) When there is a complete ascending mesocolon; (ii) When the abdominal walls are lax; (iii) When the colon is heavy with retained contents; (iv) When the patient is in the erect posture.

The structures which have been affected by traction of the mobile ascending colon in our series of cases have been: (*a*) The superior mesenteric artery, causing obstruction to the third stage of the duodenum; (*b*) The second stage of the duodenum; (*c*) The gall-bladder; (*d*) The pyloric end of the stomach; (*e*) The right kidney.

a. Traction on the Superior Mesenteric Artery—Chronic Gastromesenteric Ileus—Chronic Duodenal Ileus (Wilkie)—Arterial Duodenal Ileus.

Though obstruction of the third stage of the duodenum by the superior mesenteric artery has been recognized since 1849, when Rokitanski described it as a possible cause of acute dilatation of the stomach, Bloodgood, in 1907, was the first to demonstrate its association with a mobile ascending colon. In September, 1921, one of us[1] published 4 cases of this association, to which we now add another 15. Of the total number of cases in this series, 19 showed a pronounced degree of duodenal dilatation caused by the superior mesenteric artery.

Fig. 460.—To show the relation of the superior mesenteric artery to the duodenum and to the lumbar spine. The dotted line indicates the pelvic brim.

The blood-vessels of the normal ascending colon lie behind the parietal peritoneum, as does that segment of the gut. When a complete primitive mesentery is present the vessels pass between its leaves and share in that degree of tension to which the mesentery is subject. The superior mesenteric artery rises opposite the 1st lumbar vertebra and crosses the third stage of the duodenum opposite the 3rd. The lumbar segment of the spine is convex forwards, and the summit of its convexity corresponds to the anterior part of the body of the 3rd lumbar vertebra. The superior mesenteric artery, therefore, has to pass forwards as well as downwards from its origin, and, after crossing the duodenum, inclines somewhat backwards (*Fig.* 460). Any tension on the artery must therefore produce obstruction of the duodenum. In the normal case no such tension exists. The mesentery of the small intestine is long enough to allow the greater part of the ileum to lie on the pelvic floor, and the jejunum rests on the ileum. If the mesentery were not long enough, tension on the vessel would be inevitable. In one case the cæcum and several feet of the ileum occupied the sac of a large inguinal hernia, and obstruction to the duodenum by the artery was demonstrated. When the ascending colon is mobile, however, its mesentery, and particularly the ileocolic artery, is not always long enough to follow the excursion of the gut without tension, and hence the tension on the ileocolic artery is communicated to the superior mesenteric trunk, and obstruction to the third stage of the duodenum results.

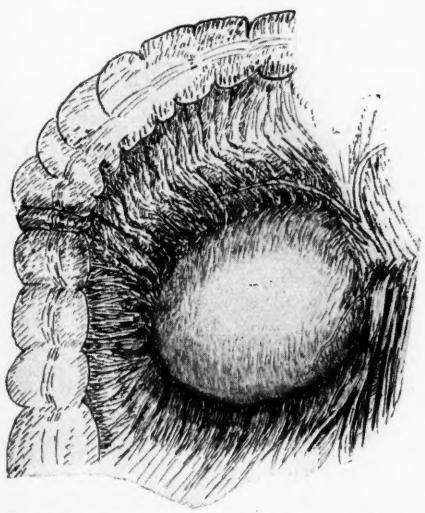


Fig. 461.—The dilated third stage of the duodenum as seen at operation.

The greater the anterior convexity of the lumbar segment of the spine, the more likely is duodenal obstruction to occur, and when this does take place, acute flexion of the thighs on the abdomen will diminish it by abolishing the anterior lumbar convexity. The more

the cæcum inclines to the back of the pelvis, the more tension is there on the superior mesenteric artery. The superior mesenteric vein is never the obstructive agent; we have repeatedly seen it passing over the dilated portion of the duodenum while the artery lay in a deep groove. This vein passes directly upwards to the portal at this point, and does not incline at all backwards: its course is at a tangent to the anterior surface of the third stage of the duodenum. Occasionally the vein and the artery are separated by a distance of an inch or more as they cross the duodenum.

Fifteen of the cases of arterial duodenal ileus seen by us have had the prolapsed type of colon. Four had the angulated type. We have but once encountered a case of arterial duodenal ileus in which the terminal ileum and cæcum did not lie in the pelvis as observed under the radioscopic screen. Parietocolic folds sometimes prevented the upper part of the ascending colon from being drawn to the middle line, and the recumbent position of the patient on the operating table sometimes brought the ileocæcal junction up, so that the colon at operation did not seem unduly mobile or prolapsed. Radioscopy occasionally reveals more than laparotomy.

The whole duodenum down to the artery was dilated so that the supramesocolic portion often presented in the wound and could even be withdrawn through it (*Fig* 461). The duodenum from its pyloric end to the crossing of the superior mesenteric artery seemed to have undergone a bucket-handle rotation.

Relation of Symptoms to Anatomical Findings.—

Case 2.—Male, age 28. Sturdy and healthy in appearance. For six years before admission, suffered from attacks of abdominal pain at irregular intervals. Each attack was characterized by the sudden onset of violent pain in the right side of the epigastrium, coming on about a quarter of an hour after food, and accompanied by vomiting, which only slightly relieved it. The interval between attacks was sometimes a few weeks, occasionally several months. Each attack came on when he was constipated, although he was not of a constipated habit, and lasted for about four days. It often started at night when he was asleep, and forced him to assume a sitting posture. He had an attack in hospital, and there was no question of the extreme severity of the pain. During the attack there was tenderness over the painful area, but no muscular rigidity. The right kidney was movable, but not markedly so, and no urinary symptoms were present. The anterior abdominal wall was of excellent tone.

X-RAY REPORT.—Normal-shaped, tonic stomach. Rapid evacuation of contents at first, but small residue retained six hours after ingestion of meal. Marked tenderness localized over pylorus and first stage of duodenum. No deformity. Ascending colon prolapsed but straight. Lateral mobility present. Positive diagnosis not justified, but there is a suggestion of ulcer at pylorus or first stage of duodenum.

OPERATION.—Stomach and biliary tract normal. The duodenum presented in the wound; it was mobile and dilated down to crossing of superior mesenteric artery. No sign of gastric or duodenal ulceration. Ascending colon was freely mobile, with complete primitive mesentery. A thin veil-like Jackson's membrane over upper part. A long appendix running up along lateral surface of ascending mesocolon. Appendicectomy—colopexy. Appendix normal. Result after two years and two months: "No return of pain or abdominal trouble of any kind; can eat and do anything."

Attacks of severe pain in the epigastrium, extending into the hypochondrium on each side, were common. Sometimes the pain was felt intensely in the back at the same or a higher level. *It was relieved by posture* in every case but one. In 12 out of 19 cases the method adopted by the patients to this end was to sit on a low stool or in bed, to draw the knees up to the chin, and clasp the legs with the arms. These patients had found this method of relief for themselves, and this it was that drew our attention to the significance of the curve of the lumbar spine. In this position the lumbar convexity was abolished, and the pressure of the thighs probably acted as a support to the prolapsed colon. Of the 7 remaining cases, 2 lay prone with a pillow or their fists under the abdomen for relief, 1 stood up and walked about. Three patients were relieved by lying down, but mentioned no special position. One patient was not relieved by any posture. Pain was increased in two cases by lying on either side: in one by lying on the right and in another by lying on the left side. In but three cases was it not increased by the supine position. *Fig.* 460 illustrates our explanation of the production or increase of pain when the patient assumes this position. The cæcum slips from the right iliac fossa

into the pelvis and tends to fall into the hollow of the sacrum, thus drawing the superior mesenteric artery more tightly across the duodenum. The pain had no relation to food in 11 cases, in 4 it came on from one to two hours after a meal, and in 1 food relieved the pain. Vomiting, copious and recurrent, was marked in 13 cases, and absent in 6. One patient had vomited nearly every day for five years. Eructations of gas were usual. All the patients complained of a sensation of distention after food, necessitating loosening of the clothing. Anything taken into the stomach when the pain was present was usually regurgitated immediately. Some of the patients were free from pain as long as the bowels moved satisfactorily. Constipation always precipitated an attack. In the majority of cases the first attack took place in childhood.

The following case shows how closely the clinical picture may resemble that of duodenal ulceration :—

Case 3.—Male, age 65. Thirteen months before admission he began to experience attacks of pain at the right side of the epigastrium and back of shoulder-blades. He described the pain as that of wind, which was relieved by eructations. Pain came on two hours after a meal and was relieved by it. He was wakened by the pain between 1 and 2 a.m. He always carried an apple about with him to eat when the pain began. The pain was always worse after a meal containing meat. These attacks lasted for a few days and recurred in a few weeks. Appetite was consistently good. Relief of pain by posture was constant. *Most relief was experienced by sitting up, hugging his knees,* and inclining to the left side. Pressure of a pillow on the abdomen when lying prone also relieved him. Lying on the right side increased the pain. Since the age of 15 he had taken a small dose of Glauber's salts every morning. Failure to secure a daily evacuation had given him abdominal discomfort when a boy. On the morning of admission to hospital he had a sudden acute pain in the epigastrium, and vomited a frothy slime. The pain was so severe that he collapsed at his work. On admission he presented the typical appearance of a perforated gastric or duodenal ulcer ; the history supported the latter diagnosis. Laparotomy revealed the duodenum enormously dilated down to the superior mesenteric artery ; no sign of duodenal ulceration could be seen. The whole ascending colon fell out of the wound. On the lesser curvature of the stomach a large indurated ulcer was found. An aperture was made in the transverse mesocolon and a penetrating ulcer of the posterior wall of the stomach was found, its floor being formed by the transverse mesocolon. This ulcer was infolded and a gastro-enterostomy performed proximal to it. There was no connection between the ulcer on the posterior wall and that on the lesser curvature. The patient made an excellent recovery.

The above case is presented in full because of the typical history of duodenal ulcer in the presence of arterial duodenal ileus and gastric ulcer, and in the absence of duodenal ulceration. [We are indebted to Sir Thomas Myles for permission to include this case in our series.]

Gastro-enterostomy had been performed previously in the two following cases :—

The first patient was well for seven years after the operation, when he developed attacks of pain in the middle of the epigastrium radiating along the left costal margin. There was no relation between the ingestion of food and the onset of pain, but if he took any food during an attack he vomited immediately. Vomiting relieved the pain, as also did the squatting posture. Radioscopy demonstrated pyloric relaxations resulting in large gushes of food from the stomach, enough passing through at a time to fill the duodenum down to the position of the superior mesenteric artery. Food also passed through the gastro-enterostomy opening. The ascending colon was prolapsed and mobile. At operation, arterial duodenal ileus was demonstrated and colopexy performed. This patient has had no further trouble since the operation fifteen months ago.

The second patient had had an anastomosis made between the jejunum and the proximal sac of an hour-glass stomach two years previously. Marked amelioration followed this operation, but there was still some pain and a sensation of fullness in the epigastrium, accompanied by flatulent eructations and nausea. The pain was relieved by the squatting position. The patient was losing weight and strength, and was becoming progressively anæmic. In addition to the epigastric symptoms she had attacks of severe pain in the right iliac fossa and a 'sinking sensation' immediately after food. The appendix was removed, but proved to be normal. Shortly after the appendicectomy we read Waugh's paper and sent for the patient. Her condition had in no way improved. *X*-ray examination showed stasis for twenty-four hours in the distal sac of the hour-glass stomach, and a mobile ascending colon of the collapsed 'concertina' type with a distended cæcum. The abdomen was again opened, and the duodenum found markedly dilated down to the crossing of the superior mesenteric artery. The ascending colon had a complete primitive mesentery. Colopexy was performed. Since this operation, nearly two years ago, the patient has steadily improved and has had no return of abdominal discomfort.

The outstanding clinical features of these cases may be summarized thus: Attacks of acute epigastric pain, not related to food, culminating in vomiting and definitely relieved by flexion of the spine. We consider the squatting posture pathognomonic of this condition. Every patient who relieved his pain in this fashion had arterial duodenal ileus. There were six patients who had none of these symptoms and yet had arterial duodenal ileus. They were operated on for symptoms referred to the right iliac fossa or for chronic constipation, and the third stage of the duodenum was found dilated down to the superior mesenteric artery. Each had a mobile ascending colon. Observation of the entire duodenum in every abdominal case is necessary in order to determine what degree of dilatation of the third stage is to be considered pathological.

Treatment of Arterial Duodenal Ileus.—Relaxation of the ileocolic artery is the essence of treatment. This may be done by keeping the cæcum up by an abdominal belt, or less effectively by diminishing the contents of the cæcum, and therefore its weight, by suitable purgatives. The rational treatment seems to be to put the cæcum and the ascending colon into their normal relation to the posterior abdominal wall and fix them there. As all our cases of arterial duodenal ileus had a mobile ascending colon, we have not performed duodenojejunostomy. *X*-ray examination after colopexy and the disappearance of the patients' symptoms have convinced us that this operation alone is curative in the majority of cases. In one case vomiting, pain in the epigastrium, and a sensation of fullness persisted after colopexy. Radioscopy showed a retention of barium in the stomach after twenty-four hours, as it was before the operation. We operated six months after the first operation with the object of performing a duodenojejunostomy, and were impressed by the fact that the third stage of the duodenum had returned to its normal size. The stomach was dilated and, as had been noted at the first operation, a small scar was present on the first stage of the duodenum. There was very slight induration, and no other sign of duodenal ulceration. It is probable, however, that a small ulcer was present. Gastro-enterostomy was performed, with excellent result.

There is no doubt that duodenojejunostomy will cure duodenal ileus. We are indebted to our colleague Sir Conway Dwyer for the opportunity of seeing the operation performed by him for this condition about eight years ago. That case has been well since. He has performed duodenojejunostomy on three other occasions with good results. If, however, prolapse of the ascending colon is the causative factor in this condition, colopexy is a simpler primary procedure.

Removal of a cause does not necessarily remove an effect, and in some cases long-standing atony of the duodenum may preclude its return to normal after the mesenteric strain is removed. Duodenojejunostomy is then indicated, as it is in cases due to shortening of the mesentery of the small intestine.[2] Murphy[3] and Wheeler[4] have each reported cases in which the obstructive agent was a peritoneal band to the right of the superior mesenteric artery.

Colopexy was performed in 14 of these cases: in 2 over two years ago, in 5 over a year and a half, in 4 over one year. Of recent cases 3 are included. Thirteen patients are well since the operation; they can eat what they like without discomfort. One patient died as the result of operation. She was a woman of 26 who had suffered from indigestion and constipation since childhood; during adolescence she became progressively anæmic. When admitted to hospital she was so weak that she had to be kept absolutely at rest for a month before even an *x*-ray examination was ventured upon. This examination demonstrated duodenal ileus. After this she was treated medically for six months, without much effect. At operation the stomach was dilated and the duodenum down to the superior mesenteric artery was greatly dilated. Colopexy was performed, and she died, apparently of shock, thirty-six hours afterwards. The post-mortem examination revealed no hæmorrhage or other abdominal catastrophe.

Of the 13 cases now well, one developed adhesions between the small intestine and the scar of the abdominal wound, and was re-operated on twice in a year for obstructive symptoms. The last operation was performed fourteen months ago. The ascending colon was found definitely fixed in a normal position. Another patient, otherwise well, complains

of stiffness and weakness in the right side a year after operation. Of the remaining 5 cases, one, a girl, age 12, was admitted as acute intussusception: laparotomy revealed duodenal ileus and a mobile ascending colon loaded with hard fæces; the colon was not fixed, as the child's condition was poor. Colonic lavage relieved the symptoms. Avoidance of constipation has kept this patient well for over two years. The same treatment was adopted in another case with similar operative findings, with similar result. In 3 cases of gastric ulcer, arterial duodenal ileus was noted, but gastro-enterostomy was performed.

There were 5 cases in which the diagnosis was established by *x*-ray examination only (*Figs.* 462, 463, and 464). One of these had a mobile colon, but the exciting cause of the duodenal obstruction was the traction of several feet of ileum prolapsed into a hernial sac. Radical cure of the hernia abolished the symptoms and *x*-ray signs of duodenal ileus. The others were treated by an abdominal belt and by the administration of liquid paraffin, which methods have somewhat relieved 3 of them, whilst the others are not improved.

FIG. 462.—*Erect.*

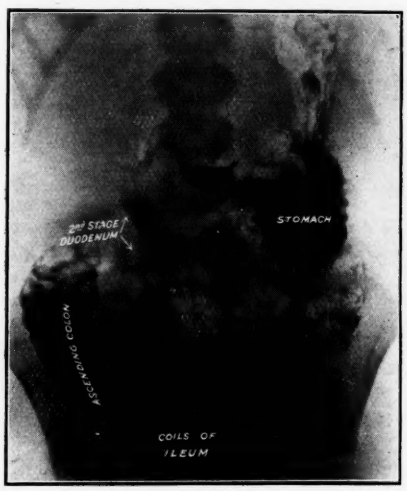


FIG. 463.—Five hours after meal.—*Erect.*

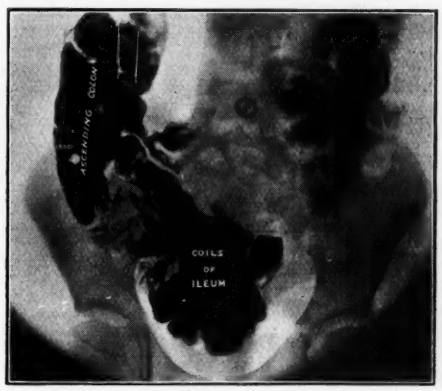


FIG. 464.—Same case—to show ascent of ascending colon. Six-and-a-half hours after meal.—*Recumbent.*

b. TRACTION ON THE DUODENUM.

The effects of traction of a mobile ascending colon on the duodenum vary according to the type of duodenum that is present. Sometimes the whole of the first stage of the duodenum can be drawn down freely, sometimes it is highly placed and fixed. A strong hepatoduodenal ligament accounts for this fixity in some cases.

We had frequently noted variations in the accessibility of the first stage of the duodenum; but it was not until we read Waugh's paper that we began to realize their significance. An investigation of these variations in infants at birth has been undertaken by Dr. C. M. West, University Anatomist, at Trinity College, Dublin; his observations,

which will be published shortly, go to prove that there is a congenital fixed type of duodenum and a congenital mobile type. These results agree with our own in the living subject, and suggest that mobility of the first stage of the duodenum is not secondary to mobility of the colon, nor are the two conditions necessarily associated (*Fig.* 465, 466, and 467).

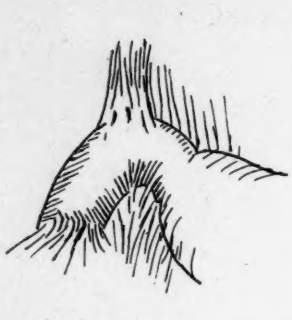


FIG. 465.—Average type.

FIG. 466.—Fixed type.

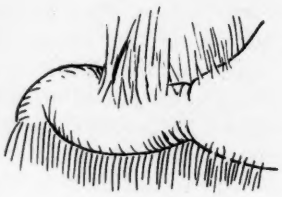
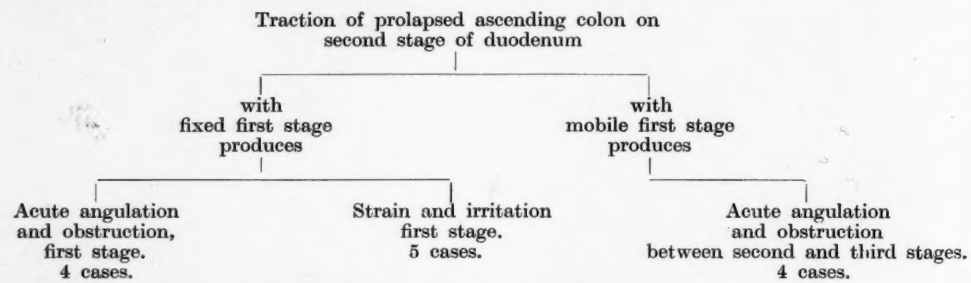


FIG. 467.—Mobile type

Both the mobile and fixed types of duodenum have been found in association with a normally fixed colon.

Second Stage of Duodenum.—In the fixed type, traction on the second stage of the duodenum produces either an acute angulation of the first stage, resulting in obstruction, or exerts a strain on the most fixed portion of the first stage, which strain may lead to local inflammatory changes.

In the mobile type, traction on the second stage draws the upper part of this portion downwards and forwards. As the third stage of the duodenum is always fixed, the descent of the first and second stages leads sooner or later to the development of an acute bend at the lower part of the second stage or at its junction with the third stage. The effects of traction on the duodenum may be tabulated thus :—


Traction of prolapsed ascending colon on second stage of duodenum
with fixed first stage produces
with mobile first stage produces
Acute angulation and obstruction, first stage. 4 cases.
Strain and irritation first stage. 5 cases.
Acute angulation and obstruction between second and third stages. 4 cases.

Acute Angulation and Obstruction, First Stage of Duodenum.—

Case 4.—Female, age 24, of healthy appearance, had an attack of acute epigastric pain and vomiting a year before. The pain came on suddenly and had no relation to food ; it radiated to the back and right shoulder, and in a few minutes culminated in copious vomiting and prostration. Inside half an hour she felt and looked well again. Exactly similar attacks recurred at intervals of anything from one to fourteen days. If she lay down immediately when the pain began, the vomiting was prevented and the pain relieved. She was always constipated, and took a purgative every morning.

RADIOSCOPY.—Dilated stomach, large six-hour residue. Tenderness over pylorus and duodenum. Caput duodeni well visualized and normal. Ascending colon prolapsed but straight, excursion of hepatic flexure 5 in., some lateral mobility. Meal reached rectum in twenty-four hours.

OPERATION.—Stomach normal, first stage of duodenum highly placed and fixed with sharp angulation, accentuated by traction on hepatic flexure of colon. No sign of ulceration. Ascending colon had complete primitive mesentery. Appendix normal. Appendicectomy and colopexy. Result, no attack since operation fourteen months ago. Radioscopy a year after : Stomach normal in size, shape, tone, and position. No six-hour residue. Excursion of hepatic flexure 3 in. ; no lateral mobility.

Acute angulation of the first stage of the duodenum with obstruction was present in three other cases. The pain was, however, related to food, coming on from half an hour to an hour and a half after a meal and relieved by recumbency or vomiting. There was no six-hour gastric residue, although the stomach was very atonic in two of the cases. The ascending colon was prolapsed in one and angulated in two. These patients have remained well after colopexy for over eighteen months.

Strain on the First Stage of the Duodenum. Duodenal Irritation.—

The anatomical findings in this group were as follows: The first stage of the duodenum was highly placed and fixed, and traction on the hepatic flexure produced an anæmic area at the point of greatest fixation. The angulation was nearly as acute as in the obstructive group just considered; the ascending colon was either of the prolapsed or angulated type; in fact it is impossible to point to any real anatomical difference between the two groups. The radioscopic and clinical findings were, however, quite different and warrant separate classification.

Case 5.—Male, age 38. He had suffered for two and a half years from attacks of pain midway between the umbilicus and the tip of the ninth costal cartilage. There was usually an interval of some weeks between the attacks. The pain came on two hours after a light meal and three hours after dinner. It lasted till the next meal, which relieved it. It awakened him about 2 a.m., and was then relieved by a biscuit. The pain was accompanied by a "rapacity rather than an appetite for food". Constipation was always associated with the attack. In the morning he felt heavy and sick. The pain was considerably relieved by lying down on his left side, and could be produced or increased by lying on his right side. From the onset of symptoms he had been under medical treatment, which included everything but the recumbent posture.

X-RAY REPORT.—Normal-shaped tonic stomach. Hypermotility present, the whole meal leaving the stomach in twenty minutes, and reaching the colon in six hours. Dropped mobile hepatic flexure. Angulation of ascending colon. *X*-ray diagnosis of duodenal irritation and mobile hepatic flexure.

OPERATIVE FINDINGS.—First stage duodenum highly placed and fixed. There was a patch of congestion at the point of greatest fixation, but no induration was present. It was slightly dilated, as also was the third stage, in which the dilatation stopped at the root of the mesentery. A firm parietocolic fold fixed the ascending colon at the junction of its lower third and upper two-thirds. Appendix normal. Appendicectomy and colopexy. Result after fifteen months: "No trouble since".

Case 6.—Female, age 29. Patient in 1916 began to suffer from pain in the epigastrium, coming on about an hour after food, and relieved by food. Though food relieved the pain it always gave her a sensation of distention. The pain was of an intense gnawing character, accompanied by nausea and eructations of gas. She was awakened by the pain at 1.30 a.m., and was then relieved by lying on her face with a pillow under the abdomen. She never vomited. When erect she could not bear the pressure of corsets. She had always been constipated. She had been treated for a few months at a time by six doctors, without relief, for five years.

No abnormality of the gastro-intestinal tract was noted on *x*-ray examination. The hepatic flexure had a vertical range of mobility of 2 in. The ascending colon was straight, and could be moved towards mid-line.

OPERATION.—Stomach normal. First stage of duodenum highly situated and fixed; it was rendered acutely angulated by traction on the mobile hepatic flexure. There was no induration or other sign of ulceration. The third stage was not dilated. The ascending colon had a complete primitive mesentery, and it seemed to be suspended from the fixed first stage of the duodenum. Owing to the high position of the latter, the ascending colon, although mobile, appeared normal on *x*-ray examination. Colopexy. Result, one year after: "Feel better than I have done for years".

There were five cases of what we have termed duodenal irritation, of which *Cases* 5 and 6 are fair examples. The general clinical aspect of the cases was as follows: Pain in the epigastrium from one to three hours after a meal, lasting to the next meal and relieved by it. Three of the patients were awakened by the pain between one and two o'clock in the morning, when two were relieved by eating something, and one by adopting the prone position. Posture had some relation to the pain in every case, but the effect was not so marked as in the other groups. One patient could produce or increase the pain by lying on the right side, another relieved it in the same manner, while the rest found some relief in the prone or supine position. Each attack was the same in the same patient, but the interval between attacks varied considerably; in two there was practically no interval.

The one thing common to each attack in each patient was that it always corresponded to a period of constipation. Four of the 5 cases showed hypertonicity and hyperperistalsis of the stomach, with rapid evacuation of its contents.

More than a year has elapsed since colopexy was performed in these cases, and in none of them have the symptoms returned. In addition to the 5 cases of duodenal irritation just recorded, 5 others with similar *x*-ray and clinical findings were observed, but were not checked by operation. One of these had been in hospital nine months previously. At that time his symptoms were all referred to the right iliac fossa. At operation an adherent appendix was removed and a strong parietocolic fold divided. This relieved all his symptoms. Soon after leaving hospital he began to develop symptoms suggestive of duodenal ulcer. When he was admitted for the second time the ascending colon was found prolapsed and straight, and the stomach showed hypermotility and rapid evacuation. The former operation had turned an angulated ascending colon into a prolapsed one, and replaced the symptoms of colonic obstruction by those of traction on the duodenum.

The significance of these cases lies in the fact that the treatment that gave them most relief was not alkalinization and not frequent meals, but an abdominal belt and liquid paraffin, two measures which tend, by supporting the ascending colon and reducing its weight, to diminish the strain exerted by it on the duodenum. There were only two cases of definite duodenal ulcer in this series. The duodenum was high and fixed, and the colon mobile. Gastro-enterostomy was performed.

Obstruction between Second and Third Stages of the Duodenum.—

Case 7.—Female, age 60. For fifteen months before admission she had suffered from attacks of pain starting in the epigastrium and radiating along the right costal margin. The attacks came on suddenly every four or five weeks, and were ushered in with pain so severe that the patient had to lie down immediately. The pain lasted about fifteen minutes and left the patient prostrate. It had no relation to food. Nausea accompanied the pain, but there was no vomiting. Immediately after the third attack jaundice appeared; this lasted about a fortnight and reappeared after each subsequent attack. The urine contained bile, and the stools were always coloured. The gall-bladder was not dilated.

X-RAY REPORT.—Normal-shaped stomach, good tone. Small barium residue seven hours and a half after meal. No deformity in the region of the pylorus. Ascending colon prolapsed, very mobile in all directions, vertical excursion of hepatic flexure 5 in. Stasis in cæcum.

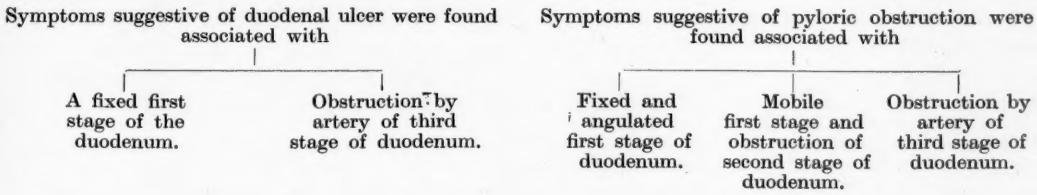
OPERATION.—Gall-bladder not dilated, no stones palpable in it or in biliary passages. Supramesocolic portion of duodenum very mobile and dilated. Slight traction on hepatic flexure brought the first stage of the duodenum *below* the level of the third. Site of obstruction was in lower part of second stage, and the duodenal deformity apparently produced obstruction of the common bile-duct. The ascending colon possessed a complete primitive mesentery. Appendicectomy and colopexy. Result after a year and nine months: "No return of pain or jaundice".

In 3 other cases operation revealed excessive mobility and dilatation of the supramesocolic portion of the duodenum without dilatation of the inframesocolic portion. These patients suffered from attacks, at irregular intervals, of epigastric pain accompanied by vomiting. The pain was considerably relieved by lying down, and the vomiting in one case was prevented by this posture. There was no jaundice. These patients had the fullest degree of mobility of the ascending colon. Colopexy was performed in each case with complete relief.

The anatomical deformity probably present in these cases, but masked by the attachment of the transverse mesocolon, was revealed in a formalin subject in the Anatomical Department of the Royal College of Surgeons in Ireland, to which Mr. A. K. Henry drew our attention. The first stage of the duodenum was very mobile, and could be lifted up readily from the posterior abdominal wall; the upper two-thirds of the second stage had a similar mobility. The supramesocolic portion as a whole lay in the horizontal plane, and could be drawn below the level of the third stage of the duodenum by gentle traction on the hepatic flexure. On dissecting away the transverse mesocolon the upper two-thirds of the second stage were seen to join the lower third at an acute angle opening downwards. The kink at this point seemed to have been sufficient to produce obstruction, for the proximal segment was dilated whilst the distal was contracted. The common bile-duct

entered the duodenum just at the bend, but there was no evidence of biliary obstruction. The ascending colon had a complete mesentery.

Remarks on Duodenal Lesions in General.—There were four separate duodenal lesions associated with mobility of the ascending colon: obstruction first stage, obstruction second stage, obstruction third stage by artery, and strain on the fixed first stage; yet there were not four separate clinical pictures, there were two. Speaking broadly, some of the patients presented symptoms suggestive of duodenal ulcer, others those suggestive of pyloric obstruction. The relation of symptoms to the anatomical condition found may be represented schematically thus :—


Symptoms suggestive of duodenal ulcer were found associated with
A fixed first stage of the duodenum.
Obstruction by artery of third stage of duodenum.
Symptoms suggestive of pyloric obstruction were found associated with
Fixed and angulated first stage of duodenum.
Mobile first stage and obstruction of second stage of duodenum.
Obstruction by artery of third stage of duodenum.

Thus the symptoms revealed not the exact *type* of lesion, but its *site*—the duodenum.

The symptoms described under 'duodenal irritation', and in *Cases* 3 and 5, sufficiently resemble those in Moynihan's classical description of duodenal ulceration to be classed as at least 'suggestive of duodenal ulcer'. Moynihan has given us the clinical picture of irritative lesions of the duodenum, and *one* of these irritative lesions is ulceration. All the cases in this series with these suggestive symptoms had something wrong with the duodenum, viz., duodenal ulcer in 1, arterial duodenal ileus in 1, and demonstrable strain on the first stage in 5. The first symptoms in one case of actual ulceration were those of peritonitis from perforation. In short, laparotomy in cases with these symptoms may not demonstrate duodenal ulcer, but it will demonstrate a duodenal lesion, if the *whole* duodenum be inspected.

c. Traction on the Gall-Bladder.

Of the patients with arterial duodenal ileus, 2 had a peritoneal fold continuous with the lesser omentum passing from the gall-bladder to the duodenum and hepatic flexure of the colon. One patient with acute suppurative cholecystitis had the same type of fold. The following case was the only one in the series in which this fold was directly responsible for symptoms :—

Case 8.—Pensioner, age 46. He had suffered at irregular intervals since 1917 from pain in the right hypochondrium radiating to the right shoulder. The pain came on suddenly, was very severe, and was accompanied by vomiting and cold perspiration. It was relieved by lying down. There was no relation to food. Pressure under the right costal margin opposite the ninth costal cartilage elicited tenderness.

Radioscopy revealed a normal gastro-intestinal tract, with no undue mobility of the ascending colon.

Operation.—A firm band continuous with the lesser omentum, passing from the gall-bladder to the duodenum and continued on to the hepatic flexure, was found. The upper third of the ascending colon had a mesentery, and traction on the colon dragged the gall-bladder downwards. The band prevented the mobile portion of the ascending colon from sagging, and so the latter had appeared normal on *x*-ray examination. The band was divided. This operation was performed ten months ago, and so far the patient has been without symptoms. Division of the band, however, has probably produced an angulated type of ascending colon.

d. Traction on the Pyloric Portion of the Stomach.

At operation it is easier to prove that the prolapsed hepatic flexure can drag on the duodenum than that it can drag on the stomach, because the duodenum is relatively fixed, whereas the stomach has a certain postural range of movement. Displacement downwards of the proximal third of the transverse colon is limited by the fixation of the normal hepatic flexure. When the flexure is not fixed, downward displacement of

the transverse colon is seen to drag on the stomach and draw its pyloric portion down as far as the gastrohepatic omentum permits. The strain of colonic traction is most marked along the lesser curvature. Variations in the length and strength of the gastrohepatic omentum probably determine whether the maximum strain falls on the stomach or on the duodenum. If the lesser omentum be long, the pyloric portion of the stomach can sink, and the weight of the ascending colon falls on the second stage of the duodenum, with the effects already noted. If it be short and strong, it acts as a suspensory ligament, not only to the pars pylorica, but also to the ascending colon and part of the transverse. Such a strain may impair the vitality of the gastric wall along the lesser curvature, and be a predisposing cause of gastric ulcer.

Case 9.—(Included by the courtesy of Sir Thomas Myles.) Male, age 52. Strong muscular development. Suffered for the last twenty years from attacks of 'roasting' pain in the epigastrium. At first the attacks occurred two or three times in the year and lasted for about three weeks, but as time went on they became increasingly frequent. He was always constipated when the pain made its appearance. For some years the pain was relieved somewhat by a hot drink, but had no other relation to food. *It was always relieved by lying down*, and never came on when he was recumbent. If he stayed in bed he could eat anything without fear of pain. Vomiting could be prevented by lying down. Medical treatment availed nothing unless he stayed in bed, when it was unnecessary. At operation a small indurated ulcer was found on the lesser curvature near the pylorus. The duodenum was normal. The ascending colon had a complete mesentery. Gastro-enterostomy was performed.

It is not probable, from the appearance of the ulcer, that it had been there for twenty years, and the characteristics of the pain never changed, nor did its relation to posture. The anatomical findings suggest that the mechanical cause of the long-standing symptoms was a factor in the causation of the short-lived ulcer.

There were 6 other cases of actual ulceration of the stomach; in all the ulcer was on the lesser curvature, and in 3 it was situated close to the incisura angularis and had produced hour-glass contraction of the stomach. The ascending colon possessed a mesentery in each case. As organic stenosis was present in all these cases, fixation of the colon was not attempted. Partial gastrectomy was performed in 2, excision of the ulcer in 1, and gastro-enterostomy in the rest. One case already cited had colopexy performed at a later date for symptoms of arterial duodenal ileus.

e. Traction on the Right Kidney.

When the hepatic flexure is in its normal relation to the anterior surface of the right kidney, it takes some part in supporting that viscus and in preventing it from slipping down the inclined plane between the last rib and the iliac crest. It is difficult to understand how nephroptosis could occur with a fixed hepatic flexure. In all the cases of movable right kidney in this series the hepatic flexure was freely mobile and was not in apposition to the kidney. When the flexure is mobile it offers no obstacle to the descent of the kidney, and the weight of the ascending colon transmitted through the peritoneal attachments to the renal fascia draws the latter downwards and thus removes a further obstacle. If the mobility of the ascending colon be the cause, or even if it be but an association of movable kidney, most of the gastro-intestinal symptoms ascribed to the latter are readily explicable. Any case might present symptoms attributable to the kidney itself, and at the same time those due to variations in the form of the ascending colon or to traction of this segment of the intestine on other structures.

Case 10.—Female, age 41, unmarried. For several years patient had suffered from attacks of flatulence and regurgitation of food. The attacks always came on when she was constipated, lasted for a day or more, and recurred at irregular intervals. Two months before admission they became more severe. When in bed at night she experienced a dull pain above the umbilicus, accompanied by flatulent eructations and a very bad taste in the mouth. She was relieved by sitting up in bed and clasping the knees. During this period she began to suffer from a dragging pain in the right loin, accompanied by frequency of micturition. This pain was relieved by lying down, and returned when she got up in the morning; it was increased by exercise. The frequency was due to polyuria. The fingers could be inserted above the upper pole of the right kidney. The stomach was dilated.

Radioscopy demonstrated retention after 6 hours in the second and third stages of the duodenum, and after 36 hours in the ascending colon, which was of the angulated type.

Operation.—Stomach dilated, duodenum dilated down to crossing of superior mesenteric artery, right kidney freely movable. Ascending colon with complete mesentery, parietocolic fold half-way up. Appendix normal. Colopexy was the only treatment adopted. Radioscopy two months after operation revealed no duodenal obstruction, and the right kidney was no longer movable. Two years after, the patient reported 'well'.

The long-standing flatulence and regurgitation of food, and the epigastric discomfort coming on so constantly during recumbency and so constantly relieved by the squatting posture, can be attributed, in the light of the cases described above, to arterial duodenal ileus; the dull dragging pain in the loin relieved by lying down, and the frequency of micturition associated with it, were doubtless due to the movable kidney.

Arterial duodenal ileus was the cause of dilatation of the stomach in another case of movable kidney. Both cases were referred to the surgical side because of the local renal symptoms.

It is significant that both patients found relief from pain in the manner described fifteen years ago by Newman in cases of movable kidney, namely by sitting up, clasping

Fig. 468.

Fig. 469.

the legs, and putting the head down on the knees. Every one of our patients who relieved his pain in this fashion had arterial duodenal ileus.

Another patient presented the symptoms of duodenal irritation in addition to those of movable kidney; at operation, traction on the mobile hepatic flexure did not affect the position of the kidney, but produced acute angulation of the first stage of the duodenum, which was highly placed and fixed. In these three cases the gastric symptoms were produced by the mobile colon and not by the movable kidney. Each patient has remained free of symptoms for more than a year after colopexy. A movable right kidney may, however, be directly responsible for duodenal obstruction, for there is no doubt that nephropexy alone removes gastric symptoms in some cases. The too frequent failures of nephropexy indicate that the primary cause of renal mobility has not been removed, or that an associated lesion has been overlooked.

There were 5 cases of movable kidney with purely renal symptoms; 1 had typical Dietl's crises; 3 had intermittent attacks of dull dragging pain in the loin accompanied by frequency of micturition; in 2 of these, hydronephrosis was demonstrated by pyelography (*Figs.* 468 and 469). The first patient has had no crisis since colopexy was performed

seventeen months ago. One case of hydronephrosis was so advanced that nephrectomy was necessary ; 1 was improved temporarily by colopexy, but a year afterwards reported that he was the same as before operation. The fourth case refused operation and the symptoms are unchanged, and the fifth was relieved by an abdominal belt. Of the 8 cases of movable kidney, 3 had well-developed abdominal muscles. None had general enteroptosis.

Lane, in 1903, attributed mobility of the right kidney to the drag of the ascending colon, and seven years later this cause of renal mobility was advocated strongly by Longyear in his book on nephrocoloptosis. This author also held that duodenal obstruction could be caused by ptosis of the ascending colon, whether the kidney was mobile or not. As far as we know, he was the first to emphasize the fact that the ascending colon was the primary cause of so many associated lesions.

6. REMARKS ON RADIOSCOPIC TECHNIQUE.

The technique adopted in the examination of our cases was as follows: The bowels were cleared by enema on the night previous to the examination, no aperient being given. At 5.30 a.m. an opaque meal was given, consisting of 3½ oz. of barium sulphate, 2 oz. of bread, and 8 oz. of milk. The patient remained in bed until the first examination six hours later. We believe that it is of the greatest importance, in determining the existence of any mechanical obstruction at the pylorus or in the duodenum, that the patient should be recumbent in the interval between the ingestion of the meal and the 6-hour examination, and in cases of this type one of us has made this procedure a routine. A 6-hour retention is abnormal, and in a recumbent patient may almost certainly be regarded as an indication of obstruction at the pylorus or in the duodenum. A hypotonic or atonic stomach may retain a large amount of the meal after six hours if the patient has been allowed to go about and carry on a normal life ; but when recumbent the same stomach may empty in the normal time, showing that no fixed obstruction is present. We first screen our patients in the erect posture, and the presence of a residue in stomach or duodenum is noted, also the position of the meal in the ileum and colon. A 6-hour retention in the stomach is regarded as abnormal, and an indication of some degree of obstruction either at the pylorus or in the duodenum. If peristalsis is proceeding, one notes whether the food passes freely through the pylorus, and, if so, whether the delay is occurring in the duodenum. If the 6-hour residue is very small, or no food remains in the stomach, a second meal is given, consisting of barium sulphate suspended in mucilage and a little water added. This fluid mixture we have found to be very suitable for the examination of the duodenum. If constriction of the third stage by the mesenteric artery is present, the accumulation of the opaque mixture proximal to this vessel can readily be seen, and in severe cases of constriction regurgitation or reverse peristalsis can be observed. In some cases we have found that the duodenum is more easily seen when in an erect position ; in others the recumbent posture is more suitable. The ability to visualize the duodenum varies greatly with different patients, and one cannot therefore lay down any hard-and-fast rule regarding the best position for examination. In those cases of duodenal ileus which we have observed radioscopically, the obstruction was plainly discernible whether the patients were standing or lying down. Plates or films are exposed if required, but in most cases we have found that the maximum amount of information may be gained by radioscopy combined with palpation. In some of our cases a 6-hour gastric residue was found but the duodenal ileus was not recognized, although found at operation. We would suggest that failure to detect this condition may have been due to pylorospasm preventing the filling and consequent visualization of the duodenum during the examination.

The examination of the colon calls for no special comment further than to say that it is best seen at the 24-hour observation as a rule, and, as already described, the measurements of the cæco-ascendens are made with the patient first standing, and then lying down.

7. GENERAL OBSERVATIONS.

The relation between symptoms and posture was so definite in these cases that it was impossible to avoid the conclusion that something inside the abdomen changed its position according to the posture of the patient, and by so doing caused or relieved the symptoms. The only structure which reacted abnormally to posture in every case was the ascending colon. Pain was associated with a definite position or deformity of the ascending colon in over 90 per cent of the cases. If the position were altered or the deformity removed, pain disappeared. If an abdominal tumour be palpated in a patient with abdominal symptoms, an attempt is made to demonstrate a connection between the tumour and the symptoms ; likewise, when the ascending colon is found deformed or mobile, it is reasonable to regard the clinical picture in relation to that abnormality.

The manner in which the symptoms are produced is primarily mechanical. Symptoms referred to the right iliac fossa were always associated with *obstruction* in the ascending colon itself. No matter how mobile the ascending colon was, the local symptoms were insignificant or absent unless angulation or collapse was present. Traction of the ascending colon on the duodenum produced some type of duodenal *obstruction* in several cases, and the symptoms were obstructive symptoms. There was a definite group of cases classed under 'duodenal irritation' in which no obstruction could be demonstrated, but in which the first stage of the duodenum was manifestly subject to strain. In short, with the exception of those present in the cases of duodenal irritation, all the gastro-intestinal symptoms were due to intermittent obstruction in the ascending colon itself or in the duodenum. Acute angulation of the ascending colon—only possible when fixation is faulty—caused obstruction in this region. Acute angulation of the duodenum and tension on the superior mesenteric artery—both produced by traction of the mobile ascending colon—were responsible for the duodenal obstruction. When the abdomen is opened under local anæsthesia, traction on a mesentery seems to be the only thing that causes pain, but such pain in our experience is referred to the back. Hurst[5] has shown that stretching or distention of the intestine causes pain, and when operating on cases of acute obstruction under local anæsthesia we have noted that pain coincides with peristalsis. Stone in the common bile-duct and ureter cause pain apart from mesenteric traction. In other words, although traction on a mesentery does cause pain, it is not the only cause, and these cases suggest that it is not the chief element in the cases of mobility of the colon.

Although an ascending mesocolon is a congenital defect, yet many years may elapse before abdominal symptoms appear, and after their onset there may be lengthy periods of immunity. Some exciting factor precipitates the owner of a mobile colon into invalidism. In the cases of *Class* 2 the exciting cause was often accumulation of fæcal masses in the ascending colon, the weight of which was thereby increased. In *Class* 1 the proximal colon sooner or later failed to compensate for the obstruction introduced in its course. A cervical rib is a congenital abnormality; yet the symptoms do not appear till some other factor—probably weakness of the musculature of the shoulder-girdle—makes its appearance.

The cases cited under 'duodenal irritation' indicate that a strain on the first stage of the duodenum is caused by the mobile colon.

Reeves, of the Mayo Clinic, has demonstrated that the arteries supplying the lesser curvature of the stomach and the first stage of the duodenum have certain peculiarities which render the blood-supply of these regions relatively deficient, and W. J. Mayo has shown that an anæmic area can be produced on the first stage of the duodenum by traction on the stomach.

In three cases with the fixed type of duodenum a similar anæmic area was produced by traction on the hepatic flexure of the colon. It is possible that the drag of the mobile colon may impair the blood-supply of the first stage of the duodenum when the patient is erect, and thus be the predisposing factor in duodenal ulceration. The relation of infection and hyperchlorhydria to such a mechanical factor would be a subject for fruitful investigation.

No physician prescribes ambulatory alkalinization in cases of duodenal ulcer; he puts the patient to bed. The recumbent position frequently takes the strain off the duodenum, and is probably as much responsible for a symptomatic cure as the raw eggs, milk, or sodium bicarbonate.

Fifty per cent of the cases here reported were males. The youngest patient was six and the oldest sixty-five.

The aim of this study was twofold: first to decide from our own experience whether the mobile ascending colon could be responsible for abdominal symptoms or not; and next, if responsible, to discover the exact anatomical manner in which it produced these symptoms. We have therefore confined ourselves to the statement of clinical states and operative findings. The good result of any operation depends on two factors: (1) That the operation is indicated; (2) That it is properly performed. Unless both factors are known, results cannot be appraised. We have not performed colopexy unless a definite anatomical connection could be traced between the mobile ascending colon and the symptoms. This connection furnished the indication for the operation, and this indication remains, whatever the results. It is with the indication for the operation that this communication deals. Some of the operations were not properly performed. In one case—the first—a carbolic swab was placed in the wound after faulty closure of the peritoneum, and caused irritation of the peritoneal surfaces; this patient was re-operated on twice for adhesions to the abdominal scar. In another case we failed to get adequate relaxation of the abdominal wall, and fixation of the colon could not be performed satisfactorily. Radioscopic examination six months afterwards showed that the colon was as mobile as ever; but whereas it had been angulated, now it was straight. The indications for colopexy can be learned by observation, the technique can be acquired by experience.

8. GENERAL TREATMENT.

These cases are orthopædic cases in the modern acceptation of that term, and the treatment necessary is orthopædic. The principles of treatment are:—

1. To make the mobile ascending colon straight.
2. To prevent it exercising traction on the structures to which it is attached.

We have failed to carry out the first principle by any non-operative method. An abdominal belt properly applied can keep the whole ascending colon up, but it cannot make it straight; angulation and collapse are accentuated.

Administration of suitable purgatives may facilitate the passage of the cæcal contents in spite of the colonic deformity, and intestinal antiseptics may partially inhibit cæcal putrefaction; but these methods are strictly palliative and applicable to the effects, not to the cause. By operation the ascending colon is first rendered straight and is then fixed in a normal position. When the symptoms are due to traction of the ascending colon on other structures, much relief may be given by non-surgical measures. The load of the ascending colon is lessened by suitable diet and laxatives, and thus its weight is diminished. An abdominal belt supports the ascending colon and prevents traction on its peritoneal connections. These measures may relieve the symptoms as a truss relieves a hernia, but they do not cure the patient. Colopexy alone does that.

Of the 76 cases in this series, 58 were operated on, colopexy being performed in 44. There were 14 cases in which the ascending colon was observed at operation but in which it was not considered advisable to fix it. These are reported in the body of the paper. Eighteen cases were not submitted to operation, but were treated by the palliative measures outlined above. All but 6 of the total number had undergone medical treatment for periods varying in length from three months to ten years, and all were referred to us by physicians.

The results may be summarized generally thus: Non-operative treatment on orthopædic lines gave better results than medical treatment, and surgical fixation of the colon better than either.

SUMMARY OF CASES.

Anatomical Findings	No. of Cases	Colopexy	Results of Colopexy	Other Operations	No Operation	Non-operative Results	
						Improved	Not Improved
Deformity of ascending colon	18	11	11 well after a year	Appendicectomy and Lane's kink 1	6	2	4
Arterial duodenal ileus	24	14	1 died, 10 well after a year, 3 well after six months, 1 slight pain and stiffness in right loin	Laparotomy 2 .. Gastro-enterostomy 3	5	3	2
Obstruction at 1st stage of duodenum	4	4	3 well after a year, 1 not improved	—	—	—	—
Strain on 1st stage of duodenum	10	5	4 well after a year, 1 slight return of old pain in spring and autumn	—	5	4	1
Obstruction at 2nd stage of duodenum	4	4	Well after a year	—	—	—	—
Traction on gall-bladder by band	1	1	Well after ten m'nths	—	—	—	—
Movable kidney ..	8	5	4 well after a year, 1 case of hydro-nephrosis not improved	Nephrectomy 1	2	1	1
Gastric ulcer ..	7	—	—	Direct treatment of ulcer and gastro-enterostomy 7	—	—	—
Total ..	76	44	1 died, 2 not improved, 1 improved but not cured	14	18	10	8

We have been exceptionally fortunate in the opportunities given us by our medical and surgical colleagues of the Richmond Hospital, who have allowed us to observe their cases and have given us full liberty in investigation. We wish to thank also all the students and nurses who submitted to radioscopic examination; without their help we could not have undertaken the inquiry. We are indebted to Dr. E. C. Smith for several of the diagrams. Professors A. F. Dixon and E. J. R. Evatt have most kindly given us every facility in their respective Anatomical Departments. The influence of Waugh's inspiring paper[6] on our work is obvious.

REFERENCES.

[1] McConnell. *Dublin Jour. Med. Sci.*, 1921, Sept.
[2] Wilkie, *Brit. Jour. Surg.*, 1921, Oct.
[3] Murphy, *Murphy's Clinics*, 1912, i, No. 2.
[4] Wheeler, *Dublin Jour. Med. Sci.* 1913, May.
[5] Hurst, *The Sensibility of the Alimentary Canal*, 1911.
[6] Waugh, *Brit. Jour. Surg.*, 1920, Jan.
Kellogg & Kellogg, *Ann. of Surg.*, 1921, May (Bibliography).

A CONTRIBUTION TO THE SURGICAL TREATMENT OF ATONIC DYSPEPSIA.

By CHARLES A. PANNETT, London.

In the continued study of cases suffering from gastric disorders, it becomes remarkable how much subjective sensations depend upon motor upset rather than upon secretory derangement. A man may go through life unaware of the fact that his stomach secretes little or no hydrochloric acid and no enzymes, but he can hardly live a completely comfortable existence if the emptying time of his stomach is delayed very much beyond the normal period of evacuation. Equally striking, when a review of a number of clinical records of gastric cases is made, is the fact that neither by the history of the illness, the results of chemical examinations, nor the changes in the appearance of the stomach cavity as visualized by Röntgen rays, can a correct diagnosis be arrived at infallibly.

A group of cases exists where, with an approximately normal secretion of juice, there is a long delay in the emptying of the stomach as shown by the barium meal. In the recrudescence and subsidence of symptoms, and in other aspects of the clinical picture, there is a very close mimicry of gastric ulcer with pyloric stenosis; yet at operation, to naked-eye inspection, no lesion can be detected in the stomach, duodenum, gall-bladder, or appendix. There is an alteration in the motor functioning of the stomach which may well be dependent upon some disturbance of the nervous mechanism by which it is controlled. On this supposition E. Bircher[1] conceived the idea of cutting off both the inhibitory impulses which travel to the general body of the stomach, and the contraction-exciting impulses which go to the pyloric ring, by section of the vagus nerves. This he did by dividing all the branches he could see near the lesser curvature, both on the anterior and posterior walls. He published a series of cases which showed remarkably favourable consequences of the operation. M. A. Latarjet[2] made a study of the nerves of the stomach, and found that the branches of the vagus contain sympathetic fibres which reach them by anastomotic paths from the cœliac plexus. He devised a slightly different procedure for section of the nerves. His investigations really show that Bircher's method in effect cuts off both vagal and sympathetic impulses. As long ago as 1886, F. Hofmeister and E. Schutz[3] demonstrated that co-ordinate peristaltic movements can take place in an excised stomach in saline solution. Two other observations in this connection are worth remembering. Stewart and Barber[4] showed that in normal dogs after sleeve resection the distal segment of the stomach exhibited more powerful and regular peristalsis, whilst W. J. Mayo[5] has observed a similar phenomenon in man after a gastric ulcer has been removed by this method. In both these instances the distal part of the stomach is almost completely cut off from nervous impulses of central origin. W. B. Cannon[6] was able to cut off nerve impulses going from the duodenum to the pylorus by making a circular incision of the duodenal wall down to the mucosa without penetrating the lumen. It seems probable that a similar operation on the stomach, at the junction of the fundus with the body, might also deprive the main part of the stomach from centrally arising nerve impulses, mainly inhibitory, and do this more effectively than by either Bircher's or Latarjet's method. This belief was tested in the patient whose clinical record is here reported. However, Barber[7] had already tested the effect of this operation in the normal stomach in animals, and found that more powerful pro- and anastaltic waves resulted in the pyloric region: in fact the effect was indistinguishable from that which he obtained by thoracic section of the vagi.

Case.—A. H., male, age 46, had suffered from intermittent attacks of pain in the right side of the epigastrium for eighteen months. The time relation to the taking of food was variable: half an hour to two hours. The pain was relieved by taking warm milk. Vomiting was a prominent feature of the attacks. Careful treatment in the medical wards had failed to bring him any relief. Other abdominal signs than some spasm of the upper recti and tenderness in the epigastric angle were wanting. The fractional test-meal was more interesting. It showed: (1) A large volume of resting contents with high acidity and a moderate amount of organic acids; (2) Marked hyperchlorhydria (hypersecretion) throughout digestion; (3) Starch present in the stomach at the end of $2\frac{2}{3}$ hours. The curve is shown in the chart (*Fig.* 470).

The radiographic examination showed that there was a dilated dropped stomach, with a large residue still left after eight hours (*Figs.* 471–475), so that organic obstruction at the pylorus seemed a safe diagnosis.

At the operation no evidence of ulcer in the stomach or duodenum was discovered. The gall-bladder had a healthy appearance, and the appendix was a normally small pale organ with no sign of inflammation or constriction. A circumcision of the stomach was made as near to the cardiac end as could conveniently be done. The incision was carried

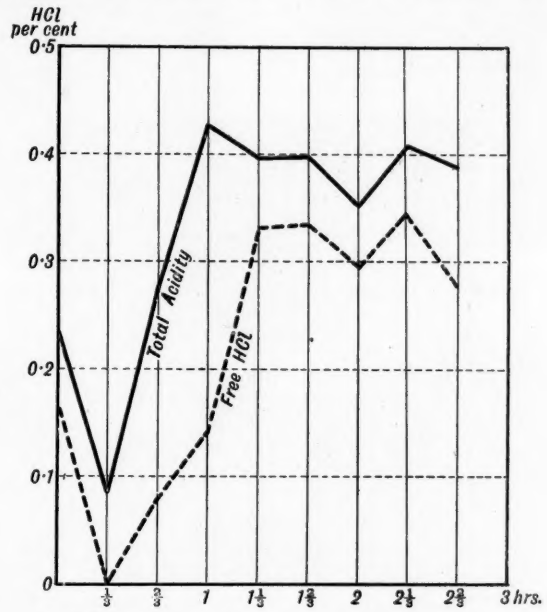


FIG. 470.—Chart showing result of the test-meal

down to the mucosa all round, except for a very small area on the greater curvature where the left gastro-epiploic artery was preserved intact; it went right across the lesser curvature, where the coronary artery was divided between ligatures. The posterior wall of the stomach was reached by traversing the great omentum, and the incision of the musculature was sewn up by a continuous catgut suture. Convalescence was quite uneventful and smooth. Three weeks after the operation, another barium meal was given, and care was taken to follow the identical technique used in the first examination. The plates show quite clearly a diminution in the size of the stomach and a much more rapid emptying: no residue in six hours in place of a large one in eight hours (*Figs.* 476–479). Even in four hours the residue is not very large. In addition, the patient affirms that all his subjective symptoms of distress after food have disappeared.

Sufficient time has not elapsed to investigate the ultimate results of this operation. A number of factors may work to vitiate the primary beneficial effects. We do not know whether a stomach deprived of all central control can permanently function efficiently, though we suspect that it can from the results of sleeve resection. Again, it is possible that the nerves may regenerate and the vicious functioning be re-established. There will

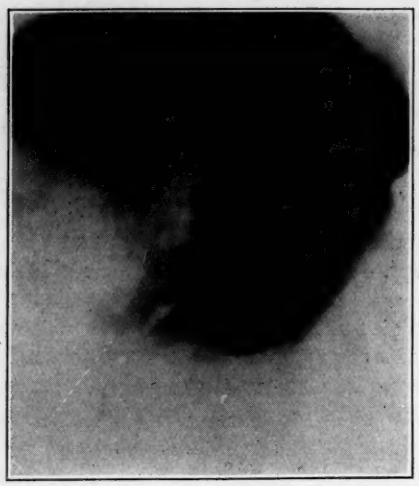


FIG. 471.—Before operation: 15-minutes plate.

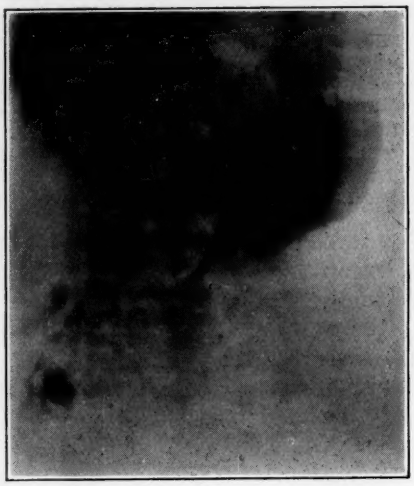


FIG. 472.—Before operation: 2-hours plate.

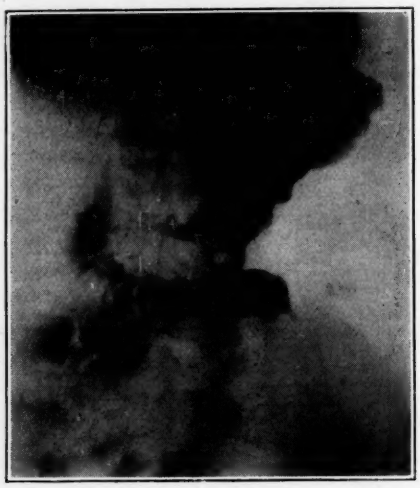


FIG. 473.—Before operation: 4-hours plate.

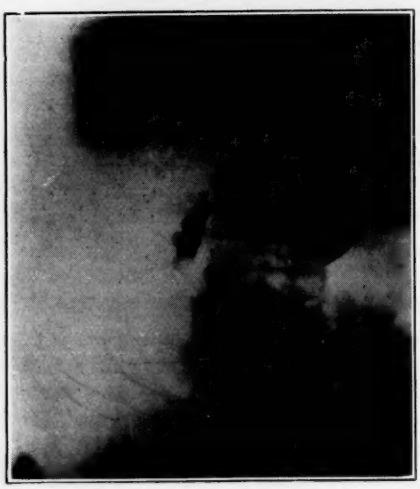


FIG. 474.—Before operation: 6-hours plate. Note that a large part of the fundus shadow is not on the plate.

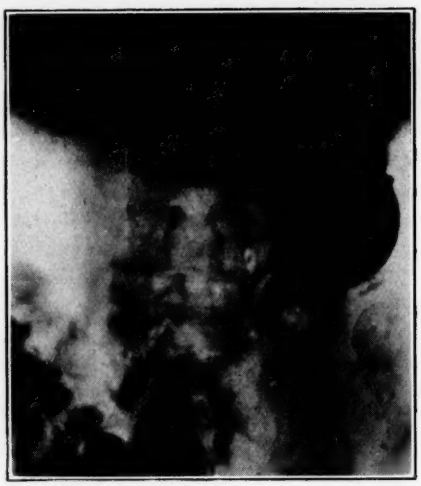


FIG. 475.—Before operation: 8-hours plate.

certainly have to be a careful selection of cases if the operation is to benefit them, for it is done on the supposition that inhibitory impulses to the wall of the stomach pass out from the central nervous system in a greater stream than normal, whilst relaxation of the pylorus is prevented by the same nervous efflux. The outstanding features in this patient were an unusually profuse acid secretion, combined with an inability of the stomach to discharge its contents.

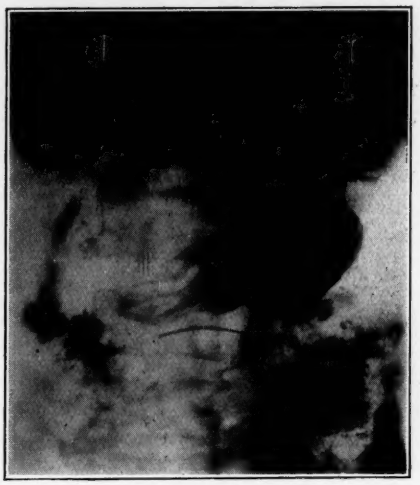


FIG. 476.—After operation: 15-minutes plate.

FIG. 477.—After operation: 2-hours plate.

FIG. 478.—After operation: 4-hours plate.

FIG. 479.—After operation: 6-hours plate.

REFERENCES.

[1] BIRCHER, E., *Schweiz. med. Woch*, 1920, l, June 17, 519.
[2] LATARJET, M. A., *Bull. de l'Acad. de Méd.*, 1922, lxxxvii, June 20, 681.
[3] HOFMEISTER, F., and SCHUTZ, E., *Arch. f. exper. Pathol. u. Pharmakol.*, 1886, xx, 1.
[4] STEWART, G. D., and BARBER, W. H., *Ann. of Surg.*, 1916, xiv, 527.
[5] MAYO, W. J., *Jour. Amer. Med. Assoc.*, 1915, Sept.
[6] CANNON, W. B., *The Mechanical Factors of Digestion*, 1911.
[7] BARBER, W. H., *Ann. of Surg.*, 1917, xvi, 672.

A CASE OF DIAPHYSIAL ACLASIS.

By A. H. SOUTHAM and R. S. PATERSON, Manchester.

The term diaphysial aclasis must be considered to be a sufficiently broad one to cover a number of disorders of growth of the diaphysis, and this atypical case of bone deformity is therefore included under this title.

The various disorders of growth affecting the skeletal system are of considerable interest owing to the diversity of forms under which they may be met with. The condition may be part of a general constitutional disease such as is seen in rickets, where practically every bone in the body may be affected, or it may be located to certain parts of the skeletal system as in achondroplasia, where only those bones formed in cartilage are affected. It is now considered that many of these diseases are due to some disturbance of function of the glands of internal secretion.

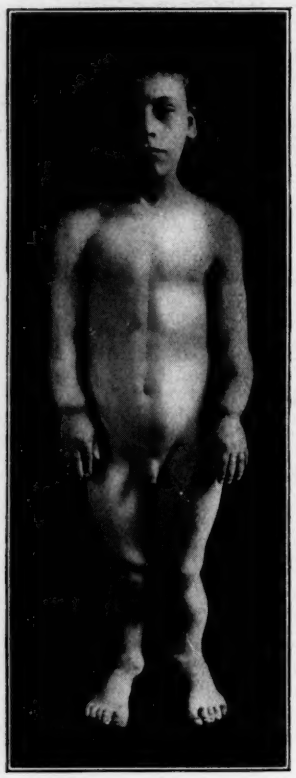


Fig. 480.

The following case appeared worthy of record on account of the unusual features of the condition, and the limitation of the bony changes almost entirely to the distal segments of the limbs.

History.—Thomas E., age 14, was brought to the out-patients' department of the Manchester Royal Infirmary by his mother for 'deformed legs'. The family history showed that the mother had borne thirteen children, eleven of whom were dead. The only other surviving son was in the army and said to show no bony deformity.

Clinical Features.—The boy walked well and appeared fairly intelligent. He was markedly stunted in height for his age, as is well shown in the photograph (*Fig.* 480), measuring only 3 ft. 7½ in., the average height at his age being 4 ft. 11 in. The forearms and legs were considerably deformed and shortened. The humerus measured 8·5 in., while the radius was only 3·5 in. in length. The enlargement of the diaphysis was very marked at the wrist. The fingers appeared stunted and thickened, the index, middle, and ring fingers all being equal in length. The femur measured 13 in., but the fibula was only 6 in. long, and the diaphyses at the ankle were markedly enlarged. The head and trunk showed no deformity or abnormality, but the sexual characteristics were distinctly in abeyance for his age. The Wassermann reaction was negative.

X-ray Appearances.—Skiagrams of almost every bone were taken, and on examination of these it was found that the changes present were confined to the forearm, leg, and some of the bones of the hand, and that the changes were bilateral. With the exception of the crests of the ilia, all the other bones were found to be comparatively normal.

The shafts of the radius and ulna were of fairly normal width but of greatly reduced length, as in achondroplasia (*Fig.* 481). Marked changes were present in the ends of the bones. The epiphyses were small and somewhat irregular, but the most pronounced changes were to be observed in the diaphyses. In all the bones affected, the diaphyses

were large and cup-shaped, with numerous bony excrescences and irregularities. The diaphysial line of dense bone, so often seen in achondroplasia, was not present; nor was the space between the epiphysis and diaphysis increased, as in rickets, although the relatively large and cupped diaphyses were suggestive of that disease.

Some of the bones of the hand showed the curious deformity of a double epiphysis; there being an epiphysis at each end of the first metacarpal bone, and one at each end of the proximal phalanx of all the fingers.

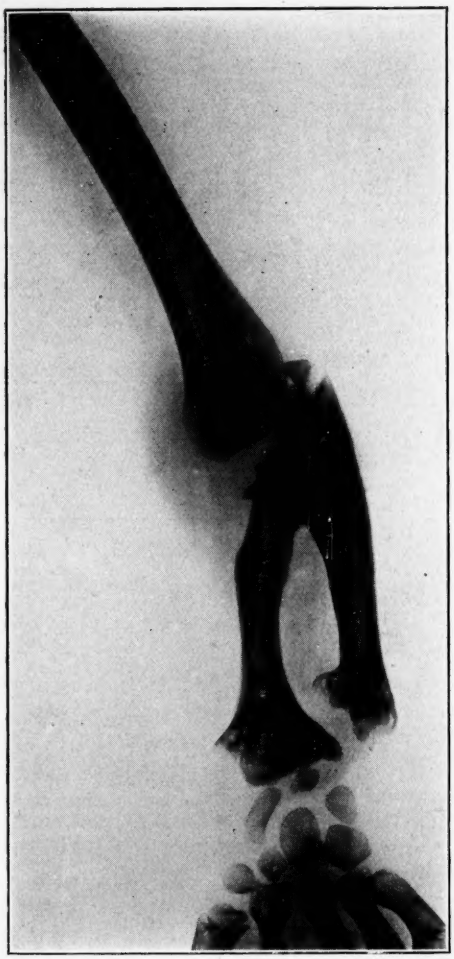


FIG. 481.

FIG. 482.

Examination of the bones of the leg (*Fig.* 482) showed changes very similar to those described above as occurring in the arm; the diaphysial exostoses at the upper end of the fibula were particularly well marked. There was relative shortening of the fibula compared with the tibia, and of the ulna compared with the radius; in the words of Keith, "the radius becomes a bent bow, the ulna serves as its taut string". Similar bony proliferations were also present at the crests of the ilia.

The skiagrams of this case have been examined by Professor Sir Arthur Keith, and he has kindly reported on them as follows:—

The *x* rays show beyond a doubt that this is a true case of diaphysial aclasis (multiple exostoses).[1] Diaphysial aclasis is really a disease or growth disorder of cartilage. So are achondroplasia and multiple enchondromata. There is a relationship between these conditions they run into each other. In this case the skull at its base shows none of the signs of achondro plasia. There are cases—as in the dachshund—where achondroplasia is confined to the limbs, and this appears to be a case of diaphysial aclasis with a leaning to achondroplasia. Diaphysial aclasis varies in its manifestations according to the date of onset.

This condition is a disorder of growth where the main disturbance falls upon the modelling of the shafts of the bones. Bones formed entirely in cartilage are free from any disorder of growth—e.g., the tarsal and carpal bones, the vertebræ, and the sternum; likewise bone formed in membrane, the bones of the cranial vault and face. The condition affects the growing ends of the bones, and is also seen along the crest of the ilium and the vertebral border of the scapula. These features are well marked in the case here described.

In achondroplasia the growth of the bones of the arm and legs is defective, the limbs appear stunted, and the stature is diminished. The bones of the trunk are normally developed as in diaphysial aclasis, as is also the vault of the skull. The base of the skull, being of cartilaginous origin, undergoes premature synostosis; this feature is absent in the case we record.

The interesting features in this case are the limitation of the abnormalities almost entirely to the distal segments of the limbs, and the reduplication of the epiphyses of the phalanges. The examination shows that it has many points in common with the condition described and designated by Sir Arthur Keith as diaphysial aclasis.

The skiagrams accompanying this article are of course composite photographs, done to show the relative appearances of the limb bones. Care was taken in obtaining them to eliminate distortion as much as possible, and the relative lengths of the bones are approximately correct as compared with the measurements taken on the subject.

REFERENCE.

[1] *Journal of Anatomy*, liv, 101.

THE CLINICAL ASPECTS OF BRANCHIAL CYSTS.

By HAMILTON BAILEY, London.

INTRODUCTION.

The study of branchial cyst is at once of morphological interest and surgical importance. In this paper the latter side of the question alone will be considered.

In May, 1855 (twenty years after Rathke's startling discovery of the existence of the branchial clefts in mammalian embryos), Langenbeck[1] described two cases of cyst of the neck in young adults. One was incised and the other treated by the insertion of a seton. In both, gruel-like material continued to discharge, which on examination was found to be rich in cholesterol. He considered these cases to be examples of persistent branchial remnants. Three years later Virchow[2] described a cyst of the neck which was excised and found to contain epidermal scales. Shortly afterwards three kindred cases were added from Volkmann's[3] clinic. From time to time other observers—notably Senn[4]—recorded cases, and gradually branchial cyst became an established clinical entity.

It is important to recognize this condition. Branchial cyst is not infrequently confounded with tuberculous cervical adenitis, as the following case summaries show :—

Case 1.—Farm labourer, age 17. For two years had a painless lump in his neck. He was told he had tuberculous glands. The swelling was aspirated seven times. Eventually a branchial cyst the size of a Tangerine orange was shelled out.

Case 2.—City girl, age 20. For two years had a swelling in left side of neck, which was aspirated five times. She was given a course of tuberculin and advised to keep in the open air. Later, a branchial cyst the size of a hen's egg was dissected out.

Case 3.—Milliner, age 27. For the past twelve years has had a lump in the neck. Ten years ago she attended a homœopathic institution and has been an occasional out-patient ever since, during which time the swelling has been aspirated no less than fifteen times. Branchial cyst, size of a Seville orange, easily dissected out.

Case 4.—Stoker, age 35. Two years ago, while at sea, a lump appeared in neck. The 'abscess' was incised by the ship's doctor. On return to home port most of his teeth were extracted. The swelling returned and has persisted. Lately it has increased in size. Large branchial cyst dissected out.

Case 5.—Female shop assistant, age 20. Eighteen months' history of a swelling in neck. Aspirated five times. 'Pus' returned sterile. Given inoculations of tuberculin and ordered oil and malt. Later, a branchial cyst the size of a hen's egg was dissected out.

Case 6.—Coal heaver, age 26. Fourteen months has had a painless swelling in neck, getting larger. He was told it was tuberculous. The swelling was aspirated four times and injected with medicated fluid. Lost his employment eight months ago because employer thought the 'neck might burst'. Branchial cyst, size of Seville orange, dissected out. (*See Fig.* 489.)

All the above cases were confirmed histologically.

It is easy to understand how this error arises. Tuberculous abscess is a far more common condition. Moreover, if an aspirating needle be thrust into a branchial cyst, the fluid withdrawn simulates tuberculous pus very closely. The specimen is naturally sent for bacteriological confirmation. But whether it be tuberculous pus or branchial fluid, the "cultures are sterile and no tubercle bacilli seen". Thus the clinician, strengthened by the bacteriological report, may begin seriously to treat a case of branchial cyst as tuberculous abscess.

The diagnosis of branchial cyst is not an academic triumph only. The possessors of these atavistic remnants are usually in the prime of life, and the stigma of tuberculosis is a heavy burden. Furthermore, if a branchial cyst be incised, there is every possibility of converting the cyst into a fistula, which continues to discharge and is the seat of recurrent attacks of inflammation, rendering subsequent removal difficult.

It will be my endeavour to show that diagnosis can usually be made with precision, and confirmed scientifically.

STRUCTURE.

The Wall and its Contents.—Gask and Wilson state that branchial cysts are lined by columnar epithelium and contain a glairy mucous fluid. That such a condition exists there can be no doubt.

The records of necropsies in the Pathological Institute of the London Hospital contain many instances of cysts in relationship with the pharynx, lined by columnar epithelium, which have given rise to no symptoms during life.

But branchial cysts lined by columnar epithelium and filled with glairy mucus seldom give rise to symptoms. I have examined the notes of 63 unpublished examples of cysts diagnosed as branchial and removed at operation. In 42 the wall of the cyst was examined histologically, and in only one was columnar epithelium found. To discover

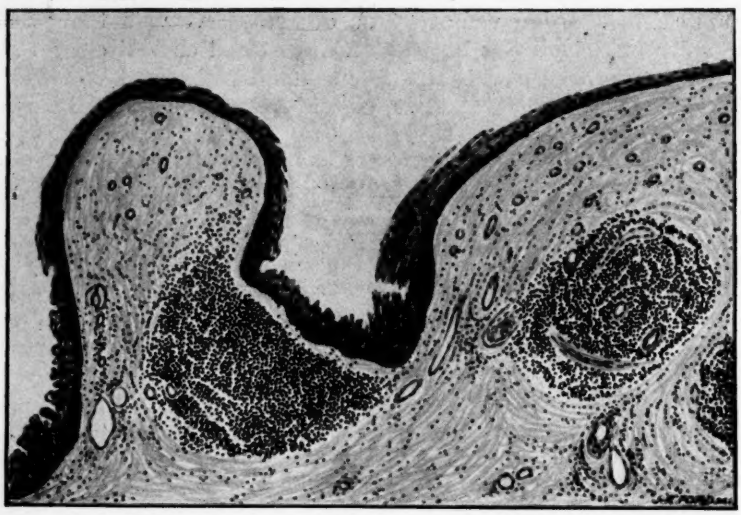


FIG. 483.—Typical section of the wall of a branchial cyst removed surgically, showing stratified squamous epithelium on a basis of lymphoid tissue.

a concrete case of an extirpated mucous branchial cyst it is necessary to search the literature—often to be rewarded by finding a cystic hygroma. Probably, therefore, the existence of the mucous branchial cyst has been established by observations in the post-mortem room rather than by investigations upon the living.

The typical lining of the cyst which gives rise to symptoms is squamous epithelium. The wall of the cyst, whether lined by squamous or columnar epithelium, is surrounded by lymphadenoid tissue (*Fig.* 483). Macroscopically the contents of the cyst lined by squamous epithelium is an opaque fluid, of such a consistency that it readily passes through an aspirating needle and often through a hypodermic needle. The similarity to tuberculous pus is striking. If, however, such branchial fluid be placed in a dish and moved to and fro, the shimmer of its lipoid contents will be noticed. The contents of the cyst will again be referred to (on page 571).

RELATIONSHIP TO SURROUNDING STRUCTURES.

The diagrams (*Figs.* 484-487) show the relationship of branchial cysts to the surrounding structures, and they are of four types:—

Type I.—Does not extend deeply. It lies at the anterior border of the sternomastoid, beneath the cervical fascia.

Type II.—Passes down to, and lies on, the great vessels. The wall has to be carefully separated from the internal jugular vein—to which it is varyingly adherent. This variety appears to be the commonest.

Type III.—Extends inwards to the lateral wall of the pharynx. From a theoretical consideration Frazer[5] states that a large branchial cyst should pass behind the carotid and in front of the vagus. However, it appears from the practical standpoint that the cyst passes between the external and the internal carotid—an observation also noted by Cunning,[6] Morrison Watson,[7] Jefferson,[8] and others. In addition, a prolongation of the cyst passes upwards as far as the lateral mass of the atlas, and even to the base of the skull.

The spinal accessory nerve is a very constant posterior relation in *Types* I and II.

It may here be stated that at operation the overlying portion of the sternomastoid has been repeatedly observed thinner and flattened out over the cyst (*Types* II and III). This point will again be referred to.

Type IV.—Is the columnar-lined cyst already considered.

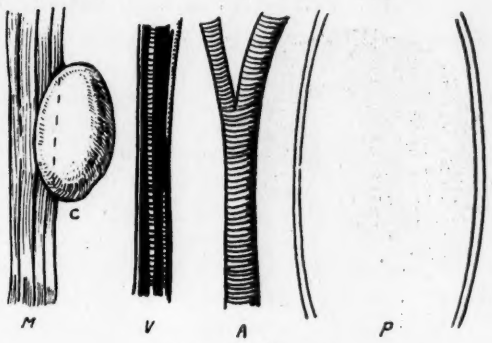


FIG. 484.—*TYPE I.*

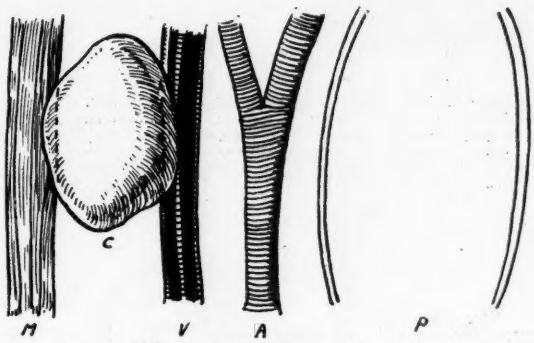


FIG. 485.—*TYPE II.*

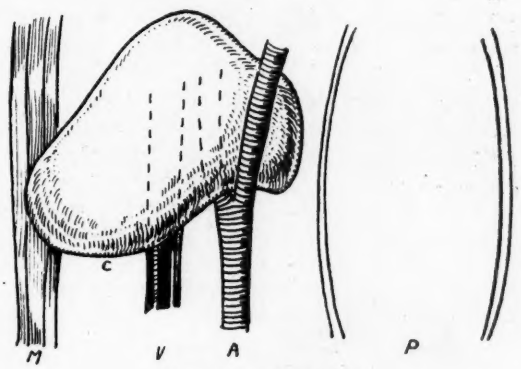


FIG. 486. - *TYPE III.*

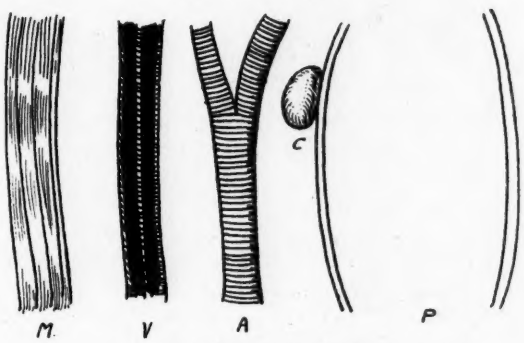


FIG. 487.—*TYPE IV.*

(M) Sternomastoid; (C) Cyst; (V) Jugular vein; (A) Carotids; (P) Pharynx.

THE PHYSICAL SIGNS ELICITED IN EIGHT CASES (1922-1923).

FIG. 488.—Age 20. Ten years' history; painless swelling, increasing in size. Swelling size of Jaffa orange. appearing around anterior border of sternomastoid; cystic, non-translucent. No cervical adenitis.

FIG. 489.—Coal heaver, age 26. Fourteen months' history; painless swelling, size of Jaffa orange, appearing around anterior border of sternomastoid; softly cystic, non-translucent. No cervical adenitis.

FIG. 490.—Age 18. Four months' history; swelling appeared after bicycle accident. Swelling size of Seville orange, appearing around posterior border of sternomastoid; cystic, non-translucent. No cervical adenitis.

FIG. 491.—Age 34. Had a small lump in neck as long as she can remember; for six years it has been increasing in size. Swelling size of Seville orange, appearing around anterior border of sternomastoid; softly cystic, non-translucent. No cervical adenitis.

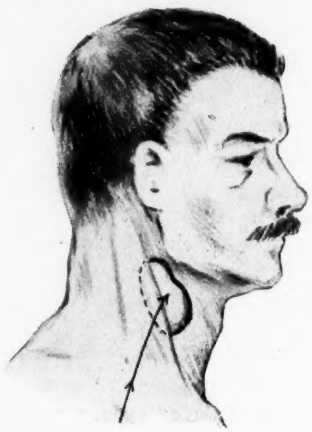


FIG. 492.—Coal heaver, age 40. Twelve months' history; painless swelling. Swelling size of hen's egg, appearing around anterior border of sternomastoid; softly cystic, non-translucent. No cervical adenitis.

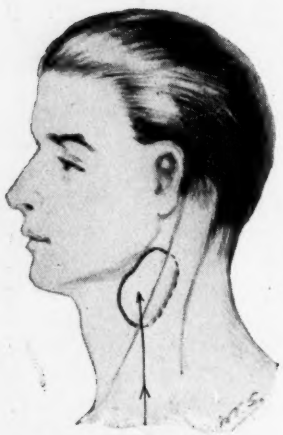


FIG. 493.—Clerk, age 19. Three years' history; painless swelling. Swelling size of Tangerine orange, appearing around anterior border of sternomastoid; cystic, non-translucent. No cervical adenitis.

FIG. 494.—Labourer, age 21. Twelve months' history; painless swelling. Swelling size of Seville orange, appearing around anterior border of sternomastoid; cystic, non-translucent. No cervical adenitis.

FIG. 495.—Age 46. Two and a quarter years' history; painless lump in neck. Lump size of hazel-nut just behind angle of jaw; movable. Diagnosed as simple parotid tumour.

COMPLICATIONS.

1. *Inflammation.*—Occasionally the non-aspirated cyst is the seat of recurrent attacks of subacute inflammation. Exceptionally suppuration occurs.

Three cases of chronic inflammation in the cyst wall were noted in the pathological reports.

2. *Fistula Formation.*—Accidental or intentional incision of the cyst is likely to result in (acquired) branchial fistula.

3. *Branchiogenetic Carcinoma.*—The existence of branchiogenetic carcinoma is, in the opinion of many, not proven. In some clinics it is a recognized diagnosis. On the other hand, no less an authority than Sir John Bland-Sutton[9] states that cancer arising in remnants of the branchial clefts is pure fiction. The whole question cannot be dealt with here. All will agree that branchiogenetic carcinoma as a diagnosis is a last refuge. It can only be entertained after a fruitless search of the mouth, nasopharynx, extralaryngeal recesses, and external auditory canal for a primary growth.

DIAGNOSIS.

Speaking generally, the making of a surgical diagnosis resolves itself into seven stages —usually not more than three or four of these will be found necessary.

1. The taking of a history and the general observation of the patient.
2. The elicitation of physical signs.
3. A mental process on the part of the surgeon whereby 1 and 2 are sifted and correlated, and a logical conclusion is drawn.
4. A differential diagnosis is entertained: also a mental process—largely one of exclusion, but reinforced when possible by further physical signs.
5. A scientific confirmatory test—usually performed by a colleague—e.g., *x*-ray, chemical, bacteriological, histological examinations.
6. The more accessible parts of the interior are rendered visible by ingeniously constructed tubes such as the cystoscope, sigmoidoscope, œsophagoscope.
7. An exploratory operation is performed.

If a diagnosis is still found wanting after the seven stages and combinations thereof have been exploited, there remains but one last court of appeal—the post-mortem room.

The seven stages, which may be termed ' the surgical crescendo ', will now be reasonably applied to the case of branchial cyst.

1. The Taking of the History: the Clinical Features.—

The complaint of the patient is the swelling; very rarely is pain a feature of the condition. In only one case was dysphagia mentioned. Not infrequently the patient states that the swelling varies in size from day to day. A few stated that the cyst became tense on occasions—and it was in these that some pain was complained of. Occasionally there is a history of injury.

Sex.—Males and females are probably equally affected, although in this series it was very slightly more common in women.

Age.—The average age at the time of seeking relief was 23. Sixty-five per cent of cases were between 17 and 30. Eight were over 40 when they came for advice.

Length of the History.—Is most frequently between 1 year and eighteen months. Eight had noticed the lump for more than 10 years.

2. The Elicitation of Physical Signs.—

Palpate the swelling.

Size.—This varies, but rarely attracts attention until it has reached the size of a hen's egg.

The Swelling is Cystic.—Occasionally, when the cyst is tense (as in the case of the breast and thyroid), it is difficult to elicit fluctuation. In these cases the lump is 'fixed' by an onlooker, and fluctuation again sought for in two planes at right angles to each other.

*Position**.—The cyst occupies a very constant anatomical position, so far as I have been able to ascertain from case histories and personal observation. It lies in relationship with the deep surface of the upper half of the sternomastoid, or some part thereof. It nearly always protrudes around the anterior border of this muscle, very occasionally around the posterior border, as *Fig.* 490 shows. Most commonly its centre is opposite the great cornu of the hyoid bone. These observations almost without exception coincide with physical signs of reported cases in the literature.

It is of great importance to determine the relationship of the swelling to the sternomastoid. It has already been pointed out that this muscle is thin and flattened out over the cyst. Consequently by mere palpation it is sometimes impossible to make out its relationships, unless the muscle be rendered taut.

Stand behind the patient. Ask him to push his chin as hard as possible against the palm of your hand. This makes the sternomastoid very tense. With the other hand palpate the sternomastoid from below (where it is normal) upwards, paying special attention to the anterior border.

Translucency.—In this series there is no record of a translucent branchial cyst. But it is possible that those rare cases springing from the pharyngeal end of the cleft, lined by columnar epithelium and filled with mucus, having attained considerable dimensions, are translucent.

3. **The Facts Collected and a Deduction made therefrom.**—"Here is a patient, age 23 who for eighteen months has had a cystic painless non-translucent swelling situated beneath the upper half of the sternomastoid, but appearing around its anterior border." It is *possible* that this is a branchial cyst. A diagnosis can never be made at this stage. It is only a guess.

4. **Differential Diagnosis.**—

i. *From Breaking-down Tuberculous Glands.*—Palpate the neck for enlarged glands. In order that no glands be overlooked, it is well to have a routine which scrutinizes every cervical lymphatic group. A useful order with a march of sequence is:—

Standing behind the patient (whose head is bent slightly forward to relax the musculature), palpate: (*a*) submental, (*b*) submaxillary, (*c*) jugular chain, (*d*) supraclavicular, (*e*) posterior triangle, (*f*) posterior auricular, (*g*) pre-auricular.

After this the possible sources of infection—ear, scalp, mouth, tonsil, etc.—are examined.

Other factors being equal: (*a*) The complete absence of cervical adenitis is in favour of the swelling being a branchial cyst; (*b*) It is unlikely, but not impossible, that a tuberculous abscess would exist for many months without the skin becoming involved.

ii. *From Cystic Hygroma* (cavernous lymphangioma).—Branchial cyst is opaque, with very few exceptions. Cystic hygroma is translucent. This sign alone is sufficient to render differential diagnosis possible. Cystic hygroma is most commonly situated in the lower half of the neck. It is usually diffuse, loculated, and first noticed in infancy.

iii. *From Solitary Lymph Cyst.*—Solitary lymph cyst is invariably translucent; it is most commonly situated in the supraclavicular triangle. This condition is probably allied to cystic hygroma. It usually, however, makes its appearance in adult life.

iv. *From a Deep-seated Venous Hæmangioma.*—This may prove a veritable trap. Differential diagnosis is rendered possible by employing the 'sign of emptying'. If the cystic swelling under consideration be a venous hæmangioma, then pressure will cause it to decrease in size; and when the pressure is removed it slowly re-fills. It must be borne in mind that it is possible for a large branchial cyst with deep ramifications to give this sign by emptying into the retropharyngeal space; therefore, while exercising this pressure, the pharynx should be watched before pronouncing the 'sign of emptying' positive.

* It is interesting to compare the position of the cyst with that of congenital branchial fistula. The orifice of the fistula (in the five cases I have been able to muster) was situated in the lower third of the neck—opposite the anterior border of the sternomastoid; the tract led upwards. In one case the fistula was bilateral.

v. *From Chronic Retropharyngeal Abscess.*—Coakley[10] (1904) was the first to recognize this possibility. His case was one of retropharyngeal swelling which had been opened three times and 'pus' evacuated. On a diagnosis of tuberculous retropharyngeal abscess, be began to operate. Opening the swelling from the pharynx a large quantity of pus-like fluid was evacuted. Introducing a finger into the cavity he was astonished to find it passed upwards to the atlas and outwards to the great vessels. A piece of smooth-lined wall was taken for section. Pathological report was : "Squamous epithelium on a basis of lymphoid tissue ".

vi. *From Lipoma.*—In all parts of the body the diagnosis of lipoma, in its usual situation, viz., superficial to the fascia, is an elementary problem. The diagnosis of *subfascial* lipoma, on the other hand, is notoriously difficult. If the differential diagnosis lies between a subfascial lipoma and branchial cyst, it can be immediately settled by the insertion of a needle ; for branchial fluid is hardly ever too thick for aspiration.

vii. *From Cystic Degeneration of a Malignant Neoplasm.*—A rapidly-growing primary or secondary malignant neoplasm in the 'branchial position', undergoing cystic degeneration, sometimes enters the question. It is, however, very unusual for a branchial cyst to make its first appearance at the carcinomatous age.

viii. *From Thyroglossal Cyst.*—Thyroglossal cyst need not necessarily be in the middle line. The levator glandulæ thyroideæ in dissecting-room subjects is most often to the left of the middle line ; a portion of the thyroglossal tract therefore is frequently on the left ala of the thyroid cartilage. Thyroglossal cyst usually bears no relationship to the sternomastoid. It never proceeds from the deep surface of that muscle. Microscopically, branchial and thyroglossal cysts are often impossible to distinguish.

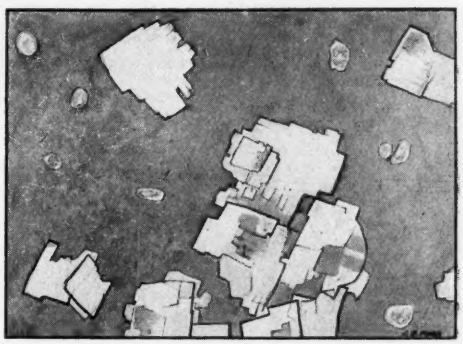


FIG. 496.—Specimen of branchial fluid, showing cholesterol and epithelial cells.

ix. *From Myxomatous Degeneration of a Mixed Parotid Tumour.*—This is a pathological curiosity. Two cases, diagnosed respectively as adenoma of parotid and cystic degeneration of mixed tumour, proved on section to be branchial cysts (evidently from the first cleft). An intraparotid branchial cyst has been described by Fredet.[11]

x. *From Aneurysm.*—This is a theoretical question only, but may be included for the sake of completeness. There is no record of a case of branchial cyst which was pulsatile from transmitted impulse of the carotids.

Having excluded these possibilities, the mental process is somewhat as follows :—

Here is a patient, age 23, who for eighteen months has had a painless non-translucent cystic swelling, which cannot be made to ' empty '. The overlying skin is quite normal. There are no enlarged cervical glands in any of the triangles of the neck. The swelling lies beneath the upper half of the sternomastoid, protruding around its anterior border, beneath the cervical fascia. The patient otherwise is healthy. Then it is *highly probable* this is a case of branchial cyst.

5. **A Scientific Confirmatory Test.**—Introduce an aspirating syringe and remove some of the fluid. Send some to the bacteriological laboratory if thought fit, but place some on a slide and look at a fresh unstained film (*Fig.* 496).

All the cases examined by this method have given a typical picture, viz., cholesterol and squamous epithelium cells. In only 1 of 8 cases examined was cholesterol absent. This could easily be explained. The patient (previously treated as a case of tuberculous abscess) had had the cyst aspirated and injected with an emulsion containing glycerol, which dissolved the cholesterol crystals. Even in this case there were abundant flattened epithelium cells.

7. An Exploratory Operation.—It has been written in a modern text-book[12] that the diagnosis of branchial cyst can seldom be made before operation. This paper is an endeavour to show that by the adoption of simple general principles it should be quite exceptional for a surgeon to fail to make a pre-operative diagnosis of branchial cyst.

In conclusion, my thanks are due to my teachers, the surgeons of the London Hospital and Liverpool Royal Infirmary, for permission to make use of their cases.

BIBLIOGRAPHY.

[1] LANGENBECK, *Arch. f. klin. Chir.*, i, 1 and 25.
[2] VIRCHOW, *Archiv, IV*, Bd. xxxv.
[3] VOLKMANN, *Arch. f. klin. Chir.*, xiv, 1.
[4] SENN, *Jour. Amer. Med. Assoc.*, 1884, iii, 197.
[5] FRAZER, J. E. S., *St. Mary's Hosp. Gaz.*, 1907, xiii, 88.
[6] CUNNING, J., *Med. Press and Circ.*, 1907, N.S., xxxiii, 226.
[7] MORRISON WATSON, *Guy's Hosp. Rep.*, 1883.
[8] JEFFERSON, J. C., *Clin. Jour.*, 1916, xiv, 424.
[9] BLAND-SUTTON, SIR JOHN, *Jour. Anat. and Physiol.*, 1886, xxi, 289; *Tumours Innocent and Malignant*, 336.
[10] COAKLEY, *Trans. Laryng. Assoc., N.Y.*, 1904, xxvi, 19.
[11] FREDET and CHEVASSU, *Bull. et Méd. Soc. Anat.*, Paris, 1902, lxxvii, 621.
[12] WARREN, R., *Text-book of Surgery*, p. 366.
BEVAN, A. D., *Surgical Clinics, Chicago*, 1920, iv, 1.
BOWLEY, SIR A., *Clin. Jour.*, 1902, xx, 225.
EISENDRATH, D., *Ann. of Surg.*, 1906, xiii, 630.
LEGG, T. P., *Med. Press and Circ.*, 1907, N.S., xxxiv, 15.
MOLE, R. H., *China Med. Jour.*, 1918, xxxii, 31.
MORTON, C. A., *Bristol Med.-Chir. Jour.*, 1911, xxix, 160.
PAUCHET, *Clin. Paris*, 1908, ii, 100.
RECLUS, *Bull. Méd. Paris*, 1906, xx, 423.

INSTRUCTIVE MISTAKE.

INJECTION OF ALCOHOL INTO THE GASSERIAN GANGLION, FOLLOWED BY WIDESPREAD CRANIAL NERVE PARALYSIS AND THE LOSS OF AN EYE.

X. Y., age 42, had suffered from trigeminal neuralgia on the right side since 1917. He had had some eight to ten injections of alcohol previous to the one which is the subject of the present note. Each injection had been followed by about three months' relief of pain. He was anxious to have an injection which would give more lasting benefit. On Dec. 22, 1921, he was given general anæsthesia (as on former occasions), and Schlosser's intrabuccal route of injection was used. The needle was felt to slip into the foramen ovale, and was pushed on for about 0·5 cm. A little clear fluid escaped from the needle, evidently cerebrospinal fluid; the needle was therefore slightly withdrawn and 1 c.c. of 90 per cent alcohol was slowly injected. The patient was sent back to bed, where he lay on his left side. The next day it was evident that he was suffering from almost complete paralysis of all the cranial nerves except the second pair. He had lost smell, taste, and hearing, he had complete ophthalmoplegia and double facial paralysis, and the movements of swallowing and articulation were impaired. Within one week it appeared that the nerves on the left side of the head were more profoundly affected than those on the right. On the right side facial and eye movements soon returned, and he could hear a little in the right ear. The left eye became the subject of ulcerative keratitis, and in spite of the utmost care the cornea perforated and the eye had to be removed.

The present condition of the cranial nerves, fifteen months after the operation, is as follows:—

I.—Loss of smell.
II.—Vision in right eye normal.
III, IV, VI.—Normal on right side.
V.—Complete anæsthesia of face and scalp as far as the vertex. Paralysis of left jaw muscles.
VII.—Normal on right. Paralysed on left.
VIII.—Right ear: Can hear sharp musical sounds—e.g., a bicycle bell—but no voice sounds. Left ear: Complete deafness.
IX.—Loss of taste.
X, XI, XII.—Normal function.

It is evident that the alcohol must have penetrated into the subarachnoid space at the base of the brain. The greater degree in which the left nerves suffered was due to the fact that the patient lay on his left side after the injection.

Three practical points are suggested by consideration of this case. First, that alcohol injection of the Gasserian ganglion is fraught with serious danger. Second, that the operation ought not to be done under general anæsthesia. Third, that if the needle after penetrating the foramen ovale draws cerebrospinal fluid, the injection of alcohol ought not to be proceeded with.

SHORT NOTES OF RARE OR OBSCURE CASES.

SPONTANEOUS RUPTURE OF A HYDRONEPHROSIS.

By W. Q. WOOD, Edinburgh.

The patient was a spare, unmarried woman of 38. She had been aware of a swelling in the left side of the abdomen for the previous ten years, which she had been informed was an enlarged spleen. She suffered some inconvenience from a constant sense of weight in the region of the swelling, and occasionally from aching pain in the left side; but in spite of this she was able to lead an active life, though she always had a desire to sit down, on account of the feeling of weight in the abdomen. Apart from occasional nocturnal frequency of micturition, she had never had any renal or bladder symptoms, and had never noticed anything abnormal about the urine.

On the night of Jan. 16, 1922, instead of going to bed in her usual sedate fashion, she jumped into bed and was immediately seized with agonizing pain in the abdomen, which was followed by vomiting. She was seen shortly afterwards by her medical attendant, and promptly sent to Chalmers' Hospital. She was seen there about an hour and a half after the onset of symptoms. She was then in the most acute distress, continually calling out and writhing about on account of the severity of the pain. She could not endure an abdominal examination, and it was impossible to make out more than a general abdominal tenderness and rigidity. The temperature was subnormal (97°) and the pulse rapid. Under general anæsthesia, it was evident that the abdomen was moderately distended. This distention appeared to be general, and no definite localized swelling could be made out. It was thought that the condition might possibly be a ruptured ovarian cyst.

Operation.—A mid-line incision was made below the umbilicus, and on opening the abdomen a large retroperitoneal swelling was found. It extended from the diaphragm above to the pelvic brim below, and was mainly on the left side. The retroperitoneal tissue, visible through the peritoneum, presented a curious œdematous appearance. A needle was introduced to ascertain, if possible, the nature of the swelling, but no fluid could be drawn off. The peritoneum was then incised on the lateral side of the descending colon, and when the colon was turned medially and the water-logged extraperitoneal tissue brushed aside, a cyst wall was discovered behind. This turned out to be the wall of a large hydronephrotic sac with a rupture towards the lateral side (*Fig.* 497). The sac was readily separated from its surroundings and removed, after the pedicle formed by the renal vessels had been secured. A tube was brought out in the loin from the space which the cyst had occupied, the parietal peritoneum repaired, and the abdomen closed.

The patient made an uneventful recovery and left hospital on the twenty-third day after operation. When seen recently (Nov. 19, 1922) she expressed herself as feeling perfectly well. She has had no urinary symptoms since the operation, and feels much fitter since being relieved of the abdominal swelling.

The hydronephrotic sac before rupture was of a large size—probably about that of an adult human cranium. The renal tissue appeared to be entirely destroyed except towards the upper end, where the wall of the sac was a little thicker than elsewhere. The outlines of the original calices could still be made out in the form of localized sacculations, but most of the specimen consisted of a smooth thin-walled cyst.

This case appears to be a rare termination of a hydronephrosis. The exciting cause of the rupture must have been the sudden increase of intra-abdominal tension when the patient jumped into bed, so that the rupture might, perhaps, be called spontaneous. Rupture from an actual trauma occasionally occurs. Nine cases were recorded by Oehne,[1] in 1907, and several have been noted since. Of Oehne's cases, 8 were operated upon. Of 3 transperitoneal nephrectomies, 2 died; of 4 lumbar, 1 died. One nephrectomy by the combined method was followed by death.

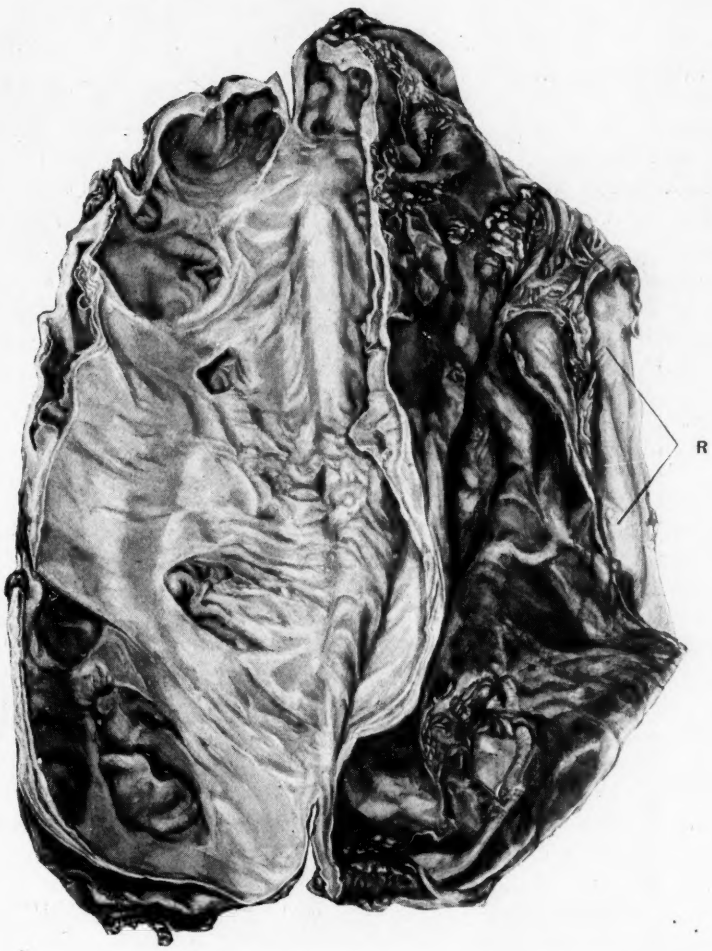


FIG. 497.—The hydronephrotic sac has been laid open from the front. The remains of the calices can be made out in the interior. The large rent (R) is seen towards the lateral border.
Reproduced from a drawing by Mrs. W. Q. Wood.

In the present case the favourable result is probably to be attributed to the absence of other injury and to the promptitude of the medical attendant in sending the patient to hospital, the patient being operated on about two hours after rupture. The only difficulty in the course of the operation arose from venous hæmorrhage, which occurred while the sac was being stripped from its surroundings. To check this, a large moist towel was packed into the lumbar region and the pedicle secured as quickly as possible.

It is interesting to note that the remaining kidney appears to be functioning perfectly.

REFERENCE.

[1]OEHNE, *Beitr. z. klin. Chir.*, 1907, lii, 715.

AN OBSCURE CERVICAL GROWTH.

By WALTER MERCER, Edinburgh.

History.—R. F. A youth, age 20, whom I saw some months ago in consultation. While on service in India on Sept. 29, 1920, the patient was taking part in a tug-of-war competition, when the rope was let go by one side and the loose end recoiled, giving him a severe blow on the right side of the neck. Beyond bruising, and pain on movement, he was comparatively well for a few days, when the pain became very much worse and he had to be admitted to hospital.

On Examination.—Oct. 20, 1920. Patient was lying flat on his back and unable to move, apparently, for fear of exciting pain. The temperature was raised and swinging, and the pulse-rate increased. He had to have everything done for him, including his feeding. All movements caused pain, particularly those of his neck and right arm, where

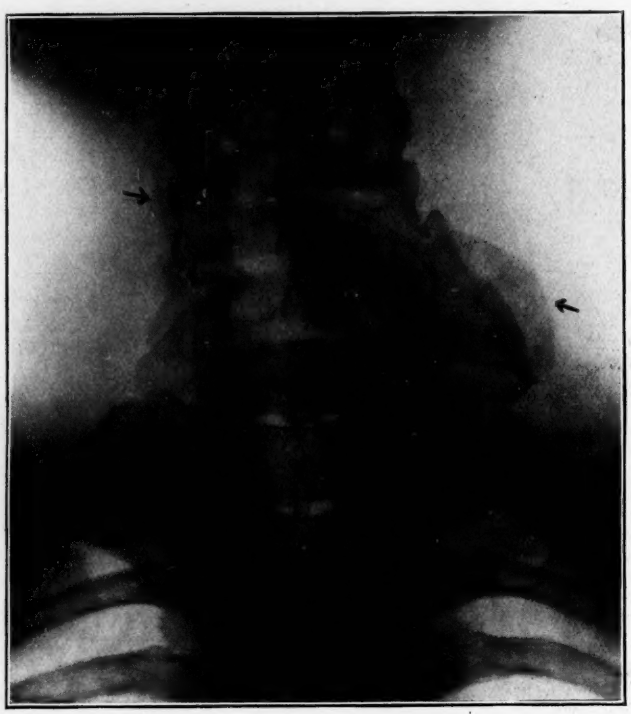


Fig. 498.—Showing the extent of bony outgrowth in the vicinity of the 5th, 6th, and 7th cervical vertebræ.

it was excruciating, and caused him to shout loudly. His neck was swollen and tender on the right side, and it was impossible to elicit any movement in it. The right arm was swollen and tender, and especially so along the lines of the nerves. There was severe neuralgic pain in these nerves, constant in character. The skin of the lower arm was tender to the touch. The right arm and face as far as the middle line were often pale in colour and covered with perspiration, while the rest of the body was quite dry. The right pupil was dilated. The lower limbs were normal, although their movements were slow and done with difficulty and he had occasional referred pain in them. The abdominal reflexes were normal, and he had control of his urine and fæces.

Nov. 15. *X* rays showed a tumour of cancellous bone attached to the right side of the 6th cervical vertebra about the size of a Tangerine orange (*Fig.* 498).

LATER HISTORY.—During the next few months the signs and symptoms progressively increased, the pain continuing severe, and the forearm and hand beginning to show signs of loss of trophic influence, the skin being glazed and the nails cracking. His pulse and temperature were little affected. Blood-count showed a polymorphonuclear leucocytosis of 12,000. He lost weight rapidly, and the continuous pain necessitated the repeated use of morphia. He was invalided home to this country with the diagnosis of tuberculous spinal caries of the 6th cervical vertebra.

As the signs and symptoms were increasing in severity and the tumour was growing in size, the case was considered later to be a sarcomatous tumour.

Up to this time little had been done in the way of treatment beyond that for the pain; but now a rigid neck support was fitted to him, and from this time onwards the symptoms improved. The pain lessened, and he was able to be got into a wheeled carriage and into the open air. In a few months the swelling in the neck became less. The blood-count improved, and the morphia was gradually stopped. The arm condition became normal, although there remained some stiffness of the joints from long-continued disuse. He was then encouraged to stand, and gradually to take a few steps.

PRESENT CONDITION.—The patient now has no pain, but there is still a swelling in the neck on the right side. This is tender to pressure. The neuritis has completely disappeared, but there remains some slight stiffness in the finger-joints and weakness in the whole right arm. The most recent skiagram—June, 1922—is reproduced, and shows the extent of the bony outgrowth in the vicinity of the 5th, 6th, and 7th cervical vertebræ. It is interesting to see that it has extended to the left side further up in the cervical vertebræ.

CONCLUSIONS.

It would seem reasonable to infer that a periostitis was set up by the injury from the rope, and that this was infected with organisms of a low virulence. The only treatment that benefited the condition was the immobilization of the neck. As soon as this was done the patient improved, and morphia was gradually diminished; his immunity increased, and he got the better of the inflammatory condition.

I have to thank Major Maurice Sinclair for permission to publish his notes of the history of the case.

INTERNAL HERNIA FOLLOWING POSTERIOR GASTRO-ENTEROSTOMY: WITH ACUTE DILATATION OF THE STOMACH AS A SEQUENCE TO REDUCTION.

BY W. TURNER WARWICK, LONDON.

ALTHOUGH the occurrence of internal hernia as a sequence of gastro-enterostomy has been recognized as a possibility almost ever since the operation was first performed, the condition is nevertheless so rare that the following case, taken from the records of the Middlesex Hospital, is of interest.

A. T., age 59, ex-policeman, was admitted to a medical ward of the Middlesex Hospital in April, 1917, complaining of chronic abdominal pain. For the previous two or three years the patient had had pain in the epigastric region, which was relieved by taking food and by vomiting. Sickness occurred at any time, with no special relation to meals. Hæmatemesis was an occasional symptom, but was not marked. The bowels were regular. The patient was a well-built man, but somewhat thin and anæmic. The appearance of the abdomen was described as scaphoid, but on clinical examination no abnormal signs were noted. A diagnosis of duodenal ulcer was made, and the patient was transferred to a surgical ward. A laparotomy confirmed the diagnosis of duodenal ulcer, and a posterior gastro-enterostomy was done. The edges of the opening in the transverse

mesocolon were not sutured to the stomach or jejunum. The patient made an uninterrupted recovery, and left the hospital three weeks later.

In May, 1919, the patient returned to hospital, stating that three months previously he had had an acute attack of pain in the pit of the stomach, which lasted some days, and that since then he had a continual dull ache on the right side of the abdomen, and across the small of his back. The pain which he had experienced previous to his operation had never recurred, and there had been no further hæmatemesis. He now suffered from flatulence, but was never sick. Since the operation he had noticed that his abdomen had begun to swell, and constipation had developed. On examination, the abdomen was now found to be very protuberant, but as the patient had gained in weight since the operation, this was thought to be due to fat. There was no visible peristalsis, nor was there any fluid in the abdominal cavity. The scar was linear and well healed. The constipation was relieved by treatment, but the pain had not entirely disappeared on his discharge three weeks later, and was thought to be functional.

In 1920 he was admitted again to the surgical wards, still complaining of pain, which he now described as dragging, in the middle of the abdomen. Since leaving hospital in 1919 he had been for some weeks an in-patient in another London hospital, but his condition had not been permanently relieved.

On examination, the abdomen was distended, but showed no rigidity or tenderness on palpation. The physical signs revealed nothing definite, but in view of the persisting symptoms an operation was decided on.

Operation.—On opening the abdomen, it was found that the whole of the small intestine with the exception of the terminal 18 inches of the ileum had herniated into the lesser sac through the opening in the mesocolon left at the previous operation. The gut was withdrawn from the sac without difficulty, and the opening closed by suturing the edges to the line of the anastomosis.

For some days after the operation the patient seemed to be progressing favourably, although vomiting of small amounts occurred at frequent intervals. The distention of the abdomen did not seem greater than before the operation. On the ninth day vomiting was more copious, and the general condition became much worse. Death occurred on the following day.

Post-mortem Examination.—This revealed an acute dilatation of the stomach. This organ filled the abdominal cavity, and the left arch of the diaphragm was considerably displaced upwards. The lower lobe of the left lung was collapsed, and the mediastinal contents were pushed over to the right. The dilatation was confined to the stomach, no part of the duodenum being affected. No sign of the old ulcer was found, nor was any other abnormal condition present in the abdomen or elsewhere.

Internal herniæ occurring after the operation of gastro-enterostomy are of two varieties.

1. The most widely recognized type takes place through an unclosed opening in the transverse mesocolon, as in the above case. In this type the whole of the small intestine may find its way through the aperture.

Moynihan[1] describes two cases illustrating the clinical sequence of events which may result from such a hernia. In the first case death occurred on the tenth day, with symptoms pointing to intestinal obstruction. A similar hernia occurred in a second case, more gradual in onset, and was operated on a year later. In this case the only prominent symptom was persistent vomiting. No mention is made here of abdominal distention. Paterson[2] states that since the adoption of the practice of suturing the edges of the mesocolon to the stomach or duodenum, no case of this complication has been recorded. A hernia similar to this in type may occur after resection of a portion of intestine, through a gap in the mesentery, when the edges are not carefully sutured. It has also been noted through congenital apertures in the mesentery and mesocolon.[3]

2. In the second variety of hernia, coils of intestine pass over the loop formed by that portion of the jejunum between the duodenojejunal flexure and the site of the gastro-

enterostomy. The possibility of its occurrence, therefore, would seem greater in the anterior operation than in the posterior modification, where the loop is so much shorter. This variety has only been described in 12 cases,[4, 5, 6] of which 7 at least followed a posterior gastro-enterostomy (5 short-loop, 2 long-loop), 3 an anterior, while in 2 the type of operation is not stated. This preponderance in the posterior modification is doubtless due to the much greater frequency with which this operation is performed.

The possibility of such a hernia also exists after anastomoses between the stomach and jejunum when a partial gastrectomy is done, although no cases have been recorded. A hernia similar in type may also occur rarely after a colostomy, round the loop of the pelvic colon, and between it and the adjacent left parietal wall.

The methods of preventing this variety of hernia suggest themselves at once, and do not call for detailed description. Thus, the space over the loop is very small in the short-loop posterior gastro-enterostomy, and can be easily closed by stitches between the jejunum and the adjacent mesocolon. The so-called 'no-loop' method of performing anterior gastro-enterostomy described by Sherren lends itself to the same treatment. When the ordinary anterior gastro-enterostomy is performed, the closure is more tedious; but suture of the loop to the mesocolon, to the great omentum, and to the stomach, will obviate the risk of hernia. The gap round the pelvic colon in colostomy can be prevented by suturing the lateral surface of the mesentery to the parietal peritoneum.

In the above case, the onset of acute dilatation of the stomach after the reduction of the hernia is of interest. The causes of acute dilatation given by Sherren[7] are: (1) Obstruction of the duodenum by the superior mesenteric artery, which crosses it (Rokitansky, Albrecht). An occasional factor in this is an adhesion of the small gut to the pelvis.[8] (2) Excessive secretion (Fagge, Henry Morris). (3) Paralysis (Campbell Thompson). (4) Septic intoxication.

Sherren also states that paralysis of the stomach is the condition regarded by most modern writers as the primary cause. In the above case the dilatation was confined to the stomach. There was no evidence at the post-mortem examination of compression of the duodenum by the superior mesenteric artery, and no sepsis was present. The explanation which seems most satisfactory is that provided by the paralytic theory. The removal of the support afforded by the crowding of the small intestine in the lesser sac may have played some part in the initiation of the condition.

REFERENCES.

[1] Moynihan, *Duodenal Ulcer*, 1912.
[2] Paterson, *Surgery of the Stomach*, 1914.
[3] Moynihan and Dobson, *Retroperitoneal Hernia*, 1906.
[4] Moscowitsch and Wilenski, *Surg. Gynecol. and Obst.* 1915, xxi, 391.
[5] Mayo and Magoun, *Arch. of Surg.* 1922, No. 22, 324.
[6] Bryan, *Surg. Gynecol. and Obst.* 1920, xxx, 82.
[7] Sherren, "Acute Dilatation of the Stomach," *Choyce's System of Surgery*, Vol. II.
[8] Spencer, *Med. Science*, 1920, iii, Nov., 145.

A CASE OF BILOCULAR GALL-BLADDER.

By BENJAMIN W. RYCROFT, Bradford.

The specimen which is the subject of this paper was taken from the following case:—

Mrs. F., a stout, florid woman, age 42, had been troubled for many years by repeated attacks of biliary colic, the first occurring in 1909, the last in May, 1922. She suffered very little discomfort during the intervals, and up to September, 1919, her health had been quite good. In the early part of October, however, whilst chasing a pig, she was seized with a severe, constricting pain in the right hypochondrium at the costal margin, which radiated to the epigastrium and round to the right flank. Jaundice supervened, and the patient was confined to bed for six weeks, during which time the pain recurred at intervals.

In the following May a severe attack was again experienced, with aggravation of all the symptoms. In November, operation was recommended.

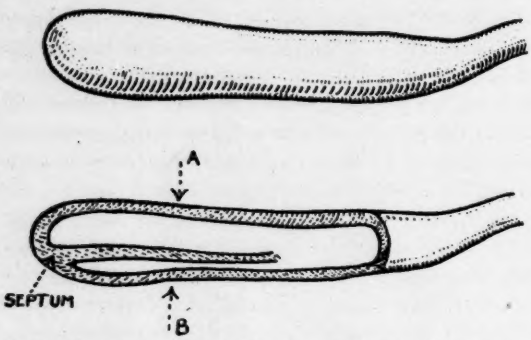


FIG. 499.—Diagrams illustrating the gall-bladder. A B shows the plane of section depicted in *Fig.* 500.

At that time there was a yellowish-green discoloration of the conjunctivæ, skin, and mucous membranes. The fæces were bulky, and putty-like in colour; the urine contained bile pigments in excess, and a trace of bile salts. Physical examination elicited deep tenderness in the right hypochondrium and epigastrium, but there was no hyperæsthesia of the skin. The recti muscles on both sides were rigid in their upper thirds. In October, 1919, the pulse-rate was 82; in November, 1922, it was 52 per minute. Constipation and pruritus of the skin had been marked in the later stages.

In November, under general anæsthesia of chloroform and ether, cholecystectomy and choledochotomy were performed. Twelve calculi were removed from the gall-bladder, and eight from the common bile-duct. The recovery of the patient was uneventful.

Examination of the Gall-bladder (*Figs.* 499, 500).—

Macroscopical Appearances.—The specimen was tubular in shape, and contracted and firm in consistency; it had very much the appearance of an appendix vermiformis. On transverse section, a septum measuring about 6½ cm. in length and ½ cm. in thickness was found, commencing in the fundus of the gall-bladder and extending along its longitudinal axis. The gall-bladder was thereby divided into two unequal loculi, the diameter of the larger being 8 mm., and of the smaller about 5 mm. A small calculus was found in the smaller loculus.

FIG. 500.—Transverse section of gall-bladder. (× 4.)

Microscopical Appearances.—Sections were cut at different levels. Each loculus was lined by columnar cells having basal nuclei. Many tubo-racemose glands were present, and those near the fundus possessed a slight degree of dilatation.

The septum intervening between the loculi was mainly composed of fibromuscular tissue, with glands dotted here and there. At its greatest thickness near the fundus it measured ½ cm.

My best thanks are due to Dr. Macnaughton, of Leicester, for permission to publish the case.

BIBLIOGRAPHY.

MOYNIHAN, *Gall-stones; their Surgical Treatment*, p. 37. 1906. (Spec. 1014, King's Coll. Museum.)
MIVART, *Comparative Anatomy*: "Complete Double Gall-bladder".

FIBROMA OF THE STOMACH.

By A. J. BLAXLAND, Norwich.

Fibromata are mentioned in certain text-books[1] as occurring in the stomach, but I can find no evidence of the report of a case. Nineteen cases of myoma or fibromyoma of the stomach, however, have been recorded.[2] Some of these were of great size—one weighing 5½ kilograms, and another being the size of a man's head. The marked microscopic resemblance between a fibroma and a fibromyoma makes it reasonable to suppose that some of these recorded cases may really have been pure fibromata.

I have recently had under my care a man from whose stomach I removed a simple solid tumour, which on microscopical examination proved to be a simple fibroma. The interesting point about the case is that, in spite of the weight and size of the tumour (which exceeded that of a billiard ball), there were no symptoms except recurrent melæna—no discomfort, otherwise, of any description.

Fig. 501.—Fibroma of stomach. *a*, Peritoneal aspect. *b*, Showing pits on the mucous surface. (× ½.)

The patient, a medical man, age 58, moderately stout, and healthy looking, had a fairly severe melæna at the end of September, 1922. A year previously he had had a severe melæna lasting several days, causing marked temporary anæmia. When 16 years old, he passed tarry motions for a week, after riding a horse. He had had no other symptoms of any sort—no indigestion, no vomiting.

Examination.—Four weeks after the last attack of melæna, the patient was obviously somewhat pale, but showed no other abnormal physical signs. There was no tenderness in the abdomen, and no swelling could be felt.

A blood-count showed a mild secondary anæmia. Red cells 4,700,000. Hæmoglobin 70 per cent. Leucocytes 7,000. Radiography after a bismuth meal showed no abnormality in the shape or motility of the stomach.

In consultation with Dr. Burton-Fanning, a provisional diagnosis of duodenal ulcer was made, and an exploratory laparotomy advised.

Operation.—On Oct. 30, a round tumour was found in the posterior wall of the stomach, its upper border being situated at the centre of the lesser curvature. An opening was made into the lesser sac, just below the greater curvature, and the stomach was turned up, exposing its posterior surface. There were no adhesions. Clamps were applied on either side of the tumour, and the stomach was opened at the lower border of the tumour, which was removed by incising round it on either side up to the lesser curvature. The gap thus caused was closed by a double layer of sutures without producing any obvious deformity of the stomach. An uneventful recovery ensued.

Description of the Specimen (*Fig.* 501).—In shape the tumour is almost spherical, its diameter measuring 2½ inches. It is slightly lobular. It is solid, but soft, giving an

impression on palpation of being cystic. Its posterior surface is covered by peritoneum, which appears to be very thin in places. The anterior surface is covered with mucous membrane, and presents four moderately deep pits (from which the hæmorrhage had presumably arisen). The tumour has a well-defined edge, and it projects equally towards the peritoneal and mucous surfaces.

MICROSCOPICAL REPORT FROM DR. CLARIDGE, NORWICH.—On examining a microscopic section of the tumour it is found to be richly cellular, composed of spindle-shaped elements arranged in interlacing bundles. The general appearance is that seen in a uterine fibroid, but muscle fibres do not appear to be present, as they do not stain characteristically by Van Gieson's method. The capsule is well marked and shows no infiltration, so that I think the tumour must be called a fibroma.

REFERENCES.

[1] KETTLE, E. H., *The Pathology of Tumours.* (H. K. Lewis & Co.)
[2] *Keen's Surgery,* vol. iii.

REVIEWS AND NOTICES OF BOOKS.

Surgery of the War. Edited by MAJOR-GENERAL SIR W. G. MACPHERSON, MAJOR-GENERAL SIR A. A. BOWLBY, MAJOR-GENERAL SIR CUTHBERT WALLACE, and COLONEL SIR CRISP ENGLISH. In two volumes. Pp. 618 and 604, with 16 and 7 coloured plates and numerous other illustrations. London: H.M. Stationery Office. 26s. each volume, post free.

THIS work has been prepared by the consulting surgeons and by some of the surgical specialists who held commissions in the Royal Army Medical Corps during the great war. Its timely appearance within a few years of the termination of the conflict has ensured that the subject of which it treats shall be described when its events are still fresh in our memory.

Volume I.—The first volume deals with general subjects affecting the surgery of the war, and indicates the development of front-line surgery and wound treatment generally, followed by chapters on wounds of the thorax and the abdomen.

The first two chapters describe the various projectiles used by the Allies and their enemies, and discusses in general terms the results of projectile action. They are written by Colonel Pilcher.

The following chapters by Colonel Cowell and Captain Fraser deal with the subject of wound shock in front-line areas and in casualty clearing stations. A very good summary of the various and conflicting theories as to the nature of shock is given. In regard to treatment, drugs are considered of no value, and in addition to the ordinary methods of warmth, administration of fluids, and sleep, the greatest importance is attached to transfusion, and it is stated that the use of gum-acacia solution has proved of enormous practical value. Major Gordon Taylor follows with a description of the methods and indications for blood transfusion, indicating the valuable results obtained by these methods, both in the treatment of wound shock and after operation.

Gas gangrene is fully described by General Sir Cuthbert Wallace, and the article is illustrated by beautiful coloured plates by Maxwell.

Sir Frederick Andrewes writes a short summary of the subject of tetanus, emphasizing the great value of the prophylactic administration of antitoxin, which reduced the incidence of the disease from over 8 per thousand to 0·2 per thousand between September, 1914, and January, 1915.

One of the most fascinating sections of the book is comprised in the four chapters which deal with surgical work in field ambulances, the development of casualty clearing stations and front-line surgery in France, wound treatment in general hospitals in France, and wound treatment in hospitals in the United Kingdom, by Colonel Max Page, General Sir Anthony Bowlby, General Sir George Makins, and Colonel Bond respectively. In these chapters we have vivid pictures of all the great difficulties, and how by organization on one hand and research on the other these difficulties were met and overcome. The story of how the treatment of grossly-infected wounds passed through many stages, both antiseptic and aseptic, is one which will remain a classic for all time.

The section on wounds of the chest and lungs is by Colonel Gask. It gives with great wealth of illustration, together with operative and mortality statistics, the history of the progress of our knowledge in this subject gained during the war. The necessity for early complete excision of infected wounds, with removal of all devitalized tissues and foreign bodies, followed by early closure of the wound, is emphasized.

The chapter on injuries to the pericardium and heart is by General Sir George Makins, and it contains a detailed account of all varieties of gunshot injury noted during the war, illustrated profusely by Maxwell's drawings. The article is accompanied by 39 case-reports from various sources of operations, both early and late, done for cardiac wounds.

The concluding chapters of the first volume, dealing with abdominal injuries, are by General Sir Cuthbert Wallace, and present a most complete account of this subject, both from the anatomical and clinical aspects. These articles are of particular value for the many tables and diagrams representing the incidence and results both of gunshot wounds and their treatment. These chapters are so full of valuable information that it is impossible to do justice to them in the space at our disposal. Expectant treatment of gunshot wounds of the abdomen has been definitely superseded by operative treatment, but the mortality still remains very high, being about 50 per cent excluding moribund cases, and about 60 per cent including these; and of all those which made an immediate recovery, about 10 per cent died subsequently, whilst only 10 per cent were fit to return to their military duties.

Volume II.—The section on wounds of the head is by Captain Trotter and Captain Wagstaffe, and embodies the generally accepted teaching in both early and late conditions. In regard to the

latter, some stress is laid upon unresolved cerebral contusion as a cause of persistent headache, and it is stated that a decompression will cure this. Another point which is perhaps open to difference of opinion refers to the necessity for closing apertures in the skull. The methods advised for this procedure are the use of celluloid plates or a piece of the tibia, whilst no mention is made of the great advantages of the cartilage graft.

The section on the face and jaw is by Major Gillies, and although it is full of useful diagrams and directions, it is disappointing, because no one injury is dealt with fully enough to give sufficient guide for actual treatment.

The sections on the spine and spinal cord are by the late Sir William Thorburn, and contain a well-balanced summary of both English and Continental views about gunshot injuries to the spine, and especially about the most important subject of the indications for operative treatment. The same author deals with the peripheral nerves in a comparatively short chapter, which makes no attempt to include the lesions of individual nerves.

The next section constitutes one of the most valuable parts of the whole work. It is concerned with injuries to the blood-vessels, and is by General Sir George Makins. It is based upon the observation of nearly 1200 cases, the notes of which were specially collected by the author. The pathological details and illustrations, and the great wealth of the clinical reports, will make this article the standard work of reference for some time to come.

The next 200 pages deal with the injuries of bones and joints, together with the general problems of so-called orthopædic treatment. Colonel Frankau treats of wounds of the joints and fractures of the upper extremity, and Colonel Webb-Johnson describes the fractures of the lower extremity. In all these articles full justice is done to the merits and advantages of the Thomas splint. The other orthopædic articles are written by General Sir Robert Jones and some of those associated with him in this work during the war. Jones himself takes the subject of stiff joints, and his advice as to treatment is on conservative lines. For example, in speaking of bony ankylosis of the knee-joint he states that "arthroplasty of the knee has no place in war surgery". This would indicate that the distinguished author does not believe in the results claimed by such workers as Putti. Major Elmslie contributes a useful article on amputations and artificial limbs.

The work concludes with chapters on injuries to the eye and ear by Colonel Lister and Major Scott. Each volume contains an index.

The editors and contributors are to be congratulated on having produced a very useful and authoritative work. The illustrations are of special value. The use of two different qualities of paper is unusual and displeasing, and the numbering of the illustrations separately in each article does not facilitate ready reference.

The Early Diagnosis of the Acute Abdomen. By ZACHARY COPE, M.D., M.S., Surgeon to Out-patients, St. Mary's Hospital, etc. Pp. 223, with 28 illustrations. 1921. London: Henry Frowde and Hodder and Stoughton. 12s. 6d. net.

THE fact that the author emphasizes repeatedly throughout the book the extreme importance of recognizing the early signs of the acute abdomen renders the work one of considerable value. Many abdominal diseases, both acute and chronic, are only recognized even to-day by what should be more accurately described as complications rather than signs and symptoms. Distention of the abdomen in peritonitis, and fæcal vomiting in intestinal obstruction, are still held by some to be signs of these diseases. They are in reality complications which should never be seen, and, if the author's advice be followed, will be more rarely met with in the future than they have been in the past. No treatment beyond operation by a competent surgeon at the earliest possible moment is discussed, and no details of operations are given. There are descriptions of the signs and symptoms and methods of diagnosis of acute abdominal catastrophes.

Several little-known signs, such as the obturator test—internal rotation of the thigh causing pain in cases of pelvic appendicitis with abscess—shoulder pain in inflammatory conditions about the diaphragm, are illustrated and explained, whilst hyperæsthesia, diminished liver dullness, and testicular pain are mentioned and their anatomical explanations given.

There are nineteen chapters in the book, the first of which is devoted to the principles of diagnosis. The all-important point of early diagnosis and the danger of delay in treatment are plainly indicated. Methods of diagnosis—the history and examination of the patient are treated very fully—illustrations of the psoas test, and the method of testing for hyperæsthesia are given.

The chapter on appendicitis is lucidly written, and many points of great importance are emphasized. Murphy's sequence of symptoms is given, and unless this occurs the practitioner should question the accuracy of his diagnosis. The absence of abdominal rigidity and even rise of temperature in pelvic abscess, and the continued acceleration of the pulse as an indication of the onset of peritonitis, are points of importance to which attention cannot too often be drawn. In the differential diagnosis of appendicitis the possibility of the presence of a stone in the right ureter should not be overlooked.

Many points in the chapter on perforated gastric and duodenal ulcers might, we think, be expressed differently. The signs and symptoms are divided into three stages: (1) Primary shock;

(2) Reaction; (3) Frank peritonitis with toxic shock. With the first of these stages we are in entire disagreement: the clinical signs and symptoms of shock are well known, and two of the most important are rapid pulse and lowered blood-pressure. We venture to say that neither of these signs is present in the early stages of perforated gastric or duodenal ulcer. In several recent cases seen by us, and according to the teaching of Moynihan which is now generally accepted, the pulse has been normal and the blood-pressure not lowered. These cases were seen within a quarter of an hour of the perforation. If the author means that there is a rapid pulse and lowered blood-pressure (clinical shock) in the first minute after the perforation, we cannot dispute this, as we have not seen cases so soon after the catastrophe. We believe that there is no stage in the perforation of a gastric or duodenal ulcer that shows the clinical signs of shock, and for this reason cannot agree that the three stages into which the author divides the signs and symptoms really exist.

In an earlier part of the work the author says that many if not most patients with a serious acute lesion of the abdomen have a normal pulse for a considerable time during the early stage. This we believe is the correct statement, and one which requires the utmost emphasis. Practitioners are inclined to say even to-day that a patient cannot have a perforation because the pulse is quite normal. We would strongly endorse the author's statement above, but equally strongly condemn the description of a primary stage of shock in perforation of a gastric or duodenal ulcer. He states that this stage may last an hour or two. With this we do not agree. We have never seen a patient in which there were the clinical signs of shock within an hour of the perforation. We maintain that there are no clinical signs of shock in the early stages of perforation; the pulse and blood-pressure are normal. This must be clearly understood, or the time for operation at which the greatest amount of good can be done will be missed.

In the chapter on acute intestinal obstruction, under the heading of diagnosis of small-gut obstruction, the author states among other signs that fæcal vomiting may occur with a flaccid, flat, and non-tender abdomen. We think that fæcal vomiting to any marked extent never takes place unless there is some distention of the abdomen. If intestinal obstruction be left until fæcal vomiting appears, the time when the success of an operation may be anticipated with some degree of certainty has already passed.

The chapter on strangulated and obstructed hernia is good; but we do not agree that it is often very difficult to differentiate between strangulated and obstructed hernia. If the hernia is tense it is strangulated, and the author rightly condemns the use of fomentations and ice-bags, and his opinion on taxis will be endorsed by all who are accustomed to deal with this condition. Strangulated inguinal, femoral, and umbilical hernias are dealt with, and the differential diagnosis of each is given.

There is a chapter on ruptured ectopic gestation, and the signs and symptoms are divided into three: (1) Signs before rupture; (2) Signs at the time of rupture; and (3) Signs after the rupture has taken place. That ectopic gestation should be diagnosed before the rupture just as appendicitis is, is the aim of the author, and with this all practitioners will agree.

Chapter XIII is devoted to cholecystitis and other causes of pain in the right upper quadrant of the abdomen. Under the heading of jaundice the author states that it is not usual in simple cholecystitis, and is not the rule even when gall-stones are present. We would say that a faint tinge of jaundice, often confined to the sclerotics, is by no means infrequent in acute cholecystitis, though it is often so faint that it will be missed unless looked for with care.

In the chapter on abdominal injuries we do not think the fact that the symptoms may be long delayed is quite sufficiently emphasized. This is especially the case in intra-abdominal rupture of the bladder. In this condition peritonitis may not be set up for many hours, and in quite a number of cases there are very few signs of shock. We do not think the absence of signs and symptoms in the early stages of severe abdominal catastrophes is sufficiently noted, as it is this absence which causes delay in operation or a false feeling of security in the mind of the practitioner. Acute pancreatitis, the acute abdomen in the tropics, diseases which may simulate the acute abdomen, acute abdominal disease with genito-urinary symptoms, are dealt with in other chapters.

On the whole the book is clearly written, and the illustrations are simple well-drawn sketches which convey to the reader exactly what the author intends they should. It is a book from which the student and practitioner can obtain a considerable amount of very valuable information, and one which, if carefully followed, will prevent undue delay in dealing with acute conditions occurring inside the abdomen.

Studien zur Anatomie und Klinik der Prostatahypertrophie. By JULIUS TANDLER and OTTO ZUCKERKANDL. Large 8vo. Pp. 130, with 121 illustrations. 1922. Berlin: Julius Springer. Price in England 23s.

THE authors of this book have worked in collaboration for seventeen years, and have published a long series of papers dealing with various phases of prostatic hypertrophy. The present volume was intended as a résumé of their work, but as Zuckerkandl unfortunately died before it was published, the survivor has brought it out as a memorial to his dead friend and colleague.

After a historical survey of the disease, the authors state that, in their opinion, prostatic hypertrophy is due to an adenomatous new growth originating in 'rudimentary prostatic glands'

situated in the upper portion of the prostatic urethra, i.e., in the portion above the verumontanum. As this growth increases in size, it compresses and flattens out the true prostatic tissue, until this simply forms a capsule round the adenomatous mass. The new growth is firmly united to the urethral mucosa, and as a consequence of this the upper part of the prostatic urethra becomes elongated by the upward development of the tumour, and its antero-posterior diameter is increased in proportion to its increase in size transversely. In fact it soon becomes a scabbard-shaped cleft between the lateral lobes of the tumour mass, the long axis of which forms a well-marked angle with the lower portion of the prostatic urethra.

The authors recognize two types of prostatic enlargement, according to the relationship of the upper end of the tumour with the vesical sphincter. In the first, the growth insinuates itself inside the sphincter, and forms a definite projection within the bladder. As this projection increases in size, it dilates the sphincter more and more, but in every case a groove corresponding to the position of the sphincter can be seen in the enucleated specimen. The intravesical projection is only covered by the mucous membrane of the bladder, and may take on various forms, e.g., a spherical tumour overhanging the urethral orifice, or a horse-shoe-shaped mass surrounding it; but no matter what its size or shape, it always takes its origin from the prostatic urethra. In the second type, the vesical sphincter is not dilated, and the adenomatous mass is entirely subvesical. The floor of the bladder is raised upwards, but the relationship of the sphincter to the other elements of the bladder base is not altered. In this type the prostatic adenoma always surrounds the urethra completely.

The effect of back-pressure on the urinary organs is next discussed. The changes in the bladder walls, trigone, seminal vesicles, etc., are minutely described; but the most interesting observation recorded is in reference to the ureter. As the prostate enlarges, the distance between the points where the ureter pierces the bladder wall and where the ejaculatory duct enters the prostate is definitely increased. The vas is put on the stretch, and pulls on and kinks the ureter at the point where they cross. Many specimens are illustrated in which the ureter is dilated and hypertrophied above this point, but of normal calibre immediately below it. In the clinical portion of the work two cystograms are reproduced, both of which show a distinct kink in the ureters just outside the bladder shadow. A short chapter is devoted to a description of inflammation arising in the enlarged prostate, and another to the development of cancer in the adenomatous nodules.

Up to this point the subject has been treated entirely from the pathological aspect, and the descriptions have been accurate, clear, and exact. There are, however, two criticisms that must occur to every reader. The first is the origin of the adenomata from 'rudimentary prostatic glands'. The existence of such glands is doubtful, and it is difficult to imagine why these glands, if they do exist, remain quiescent for the greater part of the patient's life, and only become pathological in old age. The French writers describe these tumours as arising from the normal urethral, in contradistinction to prostatic, glands; and this explanation appears to be more reasonable. The second criticism is that it is a pity the changes in the kidney due to prostatic obstruction are not described. The only change mentioned is that the 'renal pelvis and calices are dilated'; but from a practical point of view these changes are the most important of all, as the patient's life usually depends on the state of his kidneys.

About a third of the work is devoted to the clinical side of the question. A good description is given of both perineal and suprapubic prostatectomy, and of the various stages of repair after operation. This is followed by a discussion of congenital diverticula of the bladder as a complication of prostatic obstruction. A case of recurrence after prostatectomy is described, which the authors consider to be due to a fresh development of adenomata in a portion of the urethral mucous membrane left behind at the time of operation, but which, from an examination of the illustrations, appears to have had its origin from a small outlying adenoma that had become detached during enucleation. The last chapter is devoted to the question of diagnosis, but is directed chiefly to the examination of the size, shape, and cystoscopic appearances of the enlarged prostate, the appearances of the bladder, and the interpretation of cystograms, etc., while both the clinical symptoms, and the examination of the renal function, are only briefly considered.

In conclusion, the illustrations are the best feature of this work. They are extraordinarily good and clear, and render the book well worth having. The pathological portion is a clear, detailed, and comprehensive description of the changes which take place in the urethra, bladder, ureters, etc., in cases of prostatic hypertrophy. The clinical portion is not nearly so good or useful, and is marred by many strange omissions. For example, there is no mention of the pre- or post-operative treatment, the question of a two-stage prostatectomy is dismissed in five lines, and the symptomatology is very inadequately discussed. There is no bibliography, and no reference is given even when statements of other authors are quoted.

La Radiothérapie Profonde. By ISER SOLOMON, Radiologiste de l'Hôpital Sainte-Antoine. Pp. 152, with 40 illustrations. 1923. Paris: Masson et Cie. Paper covers, 9 fcs. net.

THIS is an excellent little book, well and clearly written. The subject matter is presented in a way that is concise and easily grasped without being dry. The style is excellent, and different methods and points of view are clearly stated without bias. The book should be carefully studied by all beginners, and will be of value also to experienced therapists.

The author points out in the opening sentence of his book that *x* rays, if applied in sufficient intensity, induce destructive changes upon all cellular tissues. He then refers to the work of Seitz and Wintz in regard to the dosage required to induce changes of a favourable kind in the treatment of various conditions. He is not in agreement with the very definite statements made in their work on the action upon cancer and other forms of tumours. As a basis for calculation the doses mentioned may be useful, but like many other workers he cannot accept without reservation the statement that 110 per cent of the erythema dose is the lethal dose for cancer.

He calls attention to the theory put forward by Regaud and Nogier of the specific biologic action of different wave lengths, and quotes the evidence in support of this theory. He contrasts it with the views expressed in Fredrich and Krong's well-known work. These workers maintain that the extremity of the biologic action would appear to be independent of the wave lengths of the incident rays. For the same dose absorbed in the mass of tissue the biologic action is the same for filtered and for unfiltered rays. The writer inclines to the latter view, but says no definite statement can yet be made.

The very important matter of the tube used in deep therapy is entered into ; several types are described. The boiling-water tube so much used until recently in Germany is discussed. The Lilienfeld tube is very favourably commented upon ; there is no doubt that this tube possesses certain advantages over the others.

The physics of radiation therapy has been exhaustively dealt with by the author ; the absorption of the rays by the filter, and the value in relation to a standard, such as water, are described. Water is then taken as a standard of comparison with the human tissues, and though the relation is not absolute, it is yet sufficiently near to enable the author to give useful indications for the calculation of the depth dose.

The very important matter of the divergence of the rays in the medium used is dealt with in a very lucid manner. The scattering of the rays in the tissues furnishes a very important factor in deep *x*-ray therapy. Valuable tables of absorption values are given. These are entirely useful to the *x*-ray therapist.

An important technical point is discussed in a lucid manner. Multiplication of the ports of entry or cross fire has been engaging the attention of workers for years ; this is briefly explained. Various methods for determining the coefficient of radio are described and their value indicated.

The apparatus used in deep therapy is fully described. Those designed in various centres are detailed, and a full explanation given of the intricate structure of the most popular types. The latest developments of the French technicians are referred to, and their advantages clearly put forth. The apparatus shown on page 47 gives an illustration of a complete installation. This indicates the great developments which have been made in this direction.

The important question of the measurement of the radiations is admirably dealt with, the various methods are outlined, and an excellent description is given of the ionization method. The ionometer designed by the author is fully described in the text, and the description is made easy by the inclusion of a number of good diagrams.

Propédeutique et Technique Urologiques. By Dr. G. Weiner, Bruxelles. Large 8vo. Pp. 492, with 153 illustrations. 1922. Paris : Masson et Cie. 40 fr.

This book is devoted entirely to descriptions of the various methods employed in the investigation of cases of diseases affecting the genital and the urinary tract.

Two chapters are devoted to descriptions of all the usual and unusual examinations of urine and genital secretions in health and disease. The next chapter deals in detail with cystoscopes, urethroscopes, bougies, and other instruments used by the urologist. After a description of the special methods of anæsthesia which may usefully be employed, and the indications for their employment, there follow chapters on the examination of each of the individual organs of the genito-urinary system. These are the most valuable part of the book, for the many variations met with in disease are well described, and many useful points in technique are given which simplify the investigation of difficult cases and aid in forming a correct diagnosis.

The earlier part of the book is equally well written, but does not contain much that will help the surgeon who specializes in this branch of surgery, and seems hardly likely to be read by others. There are some useful diagrams.

The Thyroid Gland. Clinics of George W. Crile and Associates. Edited by Amy F. Rowland. Pp. 288, with 106 illustrations. 1922. Philadelphia and London : W. B. Saunders Co. 24s. net.

A series of papers on various problems connected with the surgery of the thyroid gland is gathered together in this volume. Each contribution is by an expert in his own line of work, and the collection contains much information of interest to every medical man, though it will be found of particular value to those who operate upon the thyroid gland.

The opening chapters, by G. W. Crile, are of a highly theoretical nature, as, for instance, that on the rôle of the adrenals in exophthalmic goitre, and the ideas contained in them are not always easy to follow. But their author disarms criticism by explaining in the introduction that the volume is an ephemeral work, and only represents the views held in the Crile clinic at the moment of publication. The opinions expressed will be subject to revision, or may even be reversed. Many subsequent chapters, however, are eminently practical and instructive. The pathology of the thyroid gland is well presented, and there is a good account of laryngeal function in relation to the thyroid gland, attention being directed to the frequent occurrence of pre-operative abductor paralysis. Intrathoracic goitre, the value of observations on the basal metabolic rate, and the possibility of preventing the development of colloid goitre are all well discussed.

Most readers, perhaps, will find the chief interest of the book to lie in the account that is given of the pre- and post-operative care of patients with exophthalmic goitre. Great attention is given to the subject of anæsthesia, and the importance of this is not exaggerated. Stress is also laid on the prevention of operative shock, methods now well known in association with the name of Crile being described.

Some account is given of the organization of the work done in the operating theatre. In the Crile clinic this is brought to a state of efficiency such as can only be attained where large numbers of similar cases are constantly being operated upon, though it may surely serve as a useful object lesson wherever surgery is practised. Every surgeon has his own preferences in the details of operative technique, and will probably find something that is not to his mind in the methods used by others; but he cannot fail to be interested in those practised by surgeons so expert as Crile and his associates; beyond this, technical procedures and their results must be seen to be properly appreciated or criticized.

As already mentioned, the claims made for this volume are modest; yet most surgeons will regard it as a valuable addition to their libraries, and those who have enjoyed the friendship of G. W. Crile will think, as they read his words, that they can almost hear again the genial tones of his voice and feel the influence of his inspiriting presence.

The New Physiology in Surgical and General Practice. By A. Rendle Short, M.D., B.Sc., F.R.C.S. Fifth edition. Crown 8vo, revised and enlarged. Pp. 330. 1922. Bristol: John Wright & Sons Ltd. 9s. 6d. net.

It is difficult to find fault with a book which, in spite of the years of the war, has run to a fifth edition since first published in 1911. Its popularity is equally great among medical practitioners and among students reading for higher degrees in medicine and surgery.

In the present edition three new chapters appear, devoted to the physiology of muscular exercise, the functions of the kidney, and the dietetic factor in the causation of appendicitis. The last-named subject depends largely on the work in which the author himself has been especially interested, and hence is more fully dealt with than is perhaps quite justifiable in the present state of our knowledge. Most of the systems of the body are included under the various chapter headings; but although the new work on the heart is adequately reviewed by Dr. C. E. K. Herapath, the lungs do not come in for special mention. The newer methods of oxygen administration, to mention one item only, would certainly be of interest to medical practitioners, few of whom have learned to get the full value from such treatment.

The references which appear at the end of every chapter are useful to those who may wish to go further into the subject discussed. After the section on tests for renal function, mention might have been made of Professor Hugh Maclean's little book, which gives a particularly clear and concise account of the subject.

A very few errors have been noticed in perusing the book, an amusing one being the conferring of a title (no doubt well-deserved!) on a well-known neurologist who has written on aphasia.

The style in which the book is written is really excellent, and the reader's interest never flags. We agree that for medical men who are keenly interested in their work, this book must be as interesting as any novel.

Mistakes and Accidents of Surgery. By Harold Burrows, C.B.E., M.B., B.S. (Lond.), F.R.C.S. Demy 8vo. Pp. 470 + viii. 1923. London: Baillière, Tindall & Cox. 10s. 6d. net.

As we know the reputation both of the author and of the publishers, we can readily believe the latter's disclaimer that publicity in the lay press was not sought by either; but the pity is that such a disclaimer should be necessary. As we glanced through the volume—and it was purposely done far more carefully than usual—the thought "What on earth was this book written for?" constantly recurred to the mind. It is impossible to regard it as a "serious scientific work" as claimed by the publishers, for if it is to be judged by this standard, the verdict can only be that it is trivial and cursory. Then again, For whom was it written? Mr. Burrows cannot have written this book for his peers, or even for those beginning the practice of surgery in the orthodox way at a large hospital. Is it for the type of would-be operator with no surgical training? If so, it

is very dangerous diet. Many of the so-called mistakes and accidents, if they could possibly occur, could only be regarded as crimes; in truth, "Mistakes and Accidents of Surgery" is a misnomer. The book is a conglomeration of physical signs, cases briefly outlined, differential diagnosis—with some mistakes and not a few points to be avoided; much of it is perfectly sound, but it is written as if it were a shorthand report of bedside remarks which had escaped the editor's blue pencil. The surface of many subjects is skimmed, and just at the point where one's interest is aroused, another subject is introduced.

We are compelled to state with regret that this book is quite unworthy of the reputation of its author.

Lawson Tait: his Life and Work. A Contribution to the History of Abdominal Surgery and Gynæcology. By W. J. STEWART MCKAY, M.B., M.Ch. Royal 8vo. Pp. 578 + xii, with 34 plates. 1922. London: Baillière, Tindall & Cox. 25s. net.

THIS readable book, which in spite of its size is light and convenient to handle, is written by Dr. Stewart McKay, of Sydney, New South Wales, who was formerly one of Lawson Tait's assistants. It is a history of the development of modern gynæcology strung upon the thread of a life of Lawson Tait. Dr. McKay is the better fitted for his task because he published in 1901 a history of ancient gynæcology. To Dr. McKay Lawson Tait was a hero, and he naturally puts forward the best side of his character and work. He shows how much surgery was indebted to a man of comparatively little culture, who was a consummate operator and a bold surgeon. In reading his life the thought constantly recurs how much more he might have done, had he been well jostled at a large public school, if he had taken advantage of his opportunities to gain a knowledge of science at the University of Edinburgh, and if he had been attached to a hospital with a medical school where he would have been subject to the daily criticism of his colleagues and the students. The good points in his character—and they were numerous—would have stood him in better stead than they did; he would have saved himself many bitter quarrels, and he would perhaps have lived longer, for he died at the early age of 54, leaving much work to be done. But he lived his own life as a pioneer in gynæcology, pointing the way towards the modern surgery of the abdomen, and practising aseptic surgery while hardly knowing more about it than that it gave him good results, and was antagonistic to the methods recommended by Lister. His work was empirical, based to a large extent on trial and error, lacking in a recognition of the broad basic principles which a better education might have enabled him to discover.

Dr. McKay has adopted a very comprehensive plan in dealing with his subject. He first examines each important part of Tait's work in detail and then gives the modern views in order to show where the original observations have been confirmed and where experience has led to the adoption of different conclusions. By following this method the history of gynæcology is told from the first removal of a diseased ovary in 1872 until the evolution of hysteromyomectomy and the work done between 1891 and 1899. Interspersed with the general history are accounts of the results arrived at by Dr. McKay himself as a practising gynæcologist in Sydney which render the book additionally valuable. In looking back over the early period of the evolution of gynæcology it is deplorable to observe how much time was wasted and how much angry feeling was aroused by simple questions of priority and by the discussions as to the length of an incision, time which would have been spent to greater advantage in the elucidation of principles and the study of results rather than of tables of mortality.

Dr. McKay concludes with an interesting account of Tait's personal habits and methods of work as he had observed them whilst acting as his assistant in Birmingham from 1891.

Guy's Hospital Reports. Vol. 71. (Vol 2. Fourth Series). No. 1. Edited by ARTHUR F. HURST, M.D. London: Henry Frowde and Hodder & Stoughton. Subscription £2 2s. 0d. per annum. Single numbers 12s. 6d. net.

GUY'S may be congratulated on their 'Reports'; they are good and stimulating.

There is one historical article on Astley Cooper by Sir Charters Symonds which contains a good picture of the state of surgery and of medical education a hundred years ago. Osman contributes a study of case-sheets of splenic anæmia and splenomegaly, together with a good bibliography. Other articles are on "Summer Diarrhœa", "Knock-Knee", and "Vicious Circle". Nicholson writes an article on "The Heteromorphoses in the Human Body" which is intriguing and suggestive of advance in our knowledge of the growth and development of cells.

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